

EFFECTS OF HIGHER CARBOHYDRATE OR
HIGHER PROTEIN DIETS WITH EXERCISE ON INDIVIDUAL
RISK FACTORS OF METABOLIC SYNDROME IN WOMEN

A Dissertation

by

BRITTANIE LYNN LOCKARD

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Chair of Committee,	Richard B. Kreider
Committee Members,	James D. Fluckey
	Steven E. Riechman
	Steven B. Smith
Head of Department,	Richard B. Kreider

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ABSTRACT

The purpose of this analysis was to determine whether following a higher protein (HP) diet for 10-weeks promotes a reduction of MetS and the individual NCEP ATP III MetS risk factors better than a higher carbohydrate (HC) diet, when combined with an exercise program. 633 women (age 46.2 ± 11.4 yrs, height 163 ± 7 cm, weight 92.7 ± 18 kg, BMI 34.8 ± 6 kg/m²) were assigned either a HP or HC diet in conjunction with 30 minutes of circuit-style exercise 3x/wk for 10-weeks. Participants consumed $1,425 \pm 355$ kcal/day while the HP group (N=371) consumed 1.14 ± 0.5 , 1.41 ± 0.7 , and 0.63 ± 0.3 g/kg/d CHO, PRO, fat and the HC group (N=292) consumed 0.78 ± 0.3 , 2.20 ± 0.7 , and 0.60 ± 0.2 g/kg/d. Participants were retrospectively categorized as apparently healthy (N=377) or metabolic syndrome (≥ 3 MetS risk factors, N=286). Body composition, anthropometrics, resting energy expenditure, lipid profiles, markers of glucose homeostasis, and fitness parameters were assessed at 0 and 10 weeks. Data were analyzed using ANOVA or MANOVA for repeated measures. The HP group experienced a greater decrease in scanned mass (HP -3.9 ± 3.5 , HC -3.0 ± 3.5 kg, $p=0.002$), fat mass (HP -3.1 ± 2.7 , HC -2.4 ± 2.8 kg, $p=0.003$), weight (HP -4.3 ± 3.6 , HC -3.2 ± 3.4 kg, $p<0.001$), and body mass index (HP -1.6 ± 1.3 , HC -1.2 ± 1.3 kg/m², $p<0.001$), and tended to experience a greater decrease in waist circumference (HP -4.0 ± 5.7 , HC -3.2 ± 5.7 cm, $p=0.07$). Individuals with MetS experienced greater decreases in weight (AH -3.6 ± 3.4 , MS -4.2 ± 3.6 kg, $p=0.054$), body mass index (AH -1.3 ± 1.3 , MS -1.6 ± 1.3 kg/m², $p=0.046$), systolic blood pressure (AH -0.5 ± 13.3 , MS -5.9 ± 16.0 mmHg, $p<0.001$), diastolic blood pressure (AH -0.4 ± 8.9 , MS -4.1 ± 10.5 mmHg, $p<0.001$), triglycerides

(AH -0.00 ± 0.47 , -0.23 ± 0.73 mmol/L, $p < 0.001$), and glucose (AH $+0.01 \pm 0.73$, MS -0.24 ± 1.19 mmol/L, $p = 0.001$) and a trend towards a greater decrease in scanned mass (AH -3.3 ± 3.5 , MS -3.8 ± 3.5 kg, $p = 0.07$) and lean mass (AH -0.56 ± 2.0 , MS -0.89 ± 2.0 kg, $p = 0.07$). Results indicate that participants following the HP diet experienced more favorable changes in body composition and triglyceride levels, and that participants with MetS have greater room for improving markers of health on a diet and exercise protocol.

DEDICATION

To my husband, Jim: who always encourages me to dream big and continuously demonstrates that when you make your dreams your goals, those dreams can be achieved. Thank you for your love and support, and for the frequent (and much needed!) reminders that, “the only way to eat an elephant is one bite at a time.”

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NOMENCLATURE

1RM	One Repetition Maximum
AH	Apparently Healthy Study Group
ANOVA	Analysis of Variance
ATP III	Adult Treatment Panel III
BMI	Body Mass Index
BP	Blood Pressure
CHO	Carbohydrate
CVD	Cardiovascular Disease
DBP	Diastolic Blood Pressure
DEXA	Dual Energy X-Ray Absorptiometry
ESNL	Exercise and Sport Nutrition Lab
FFM	Fat Free Mass
FM	Fat Mass
HC	Higher Carbohydrate Diet
HDL	High Density Lipoprotein
HP	Higher Protein Diet
IDF	International Diabetes Foundation
LDL	Low Density Lipoprotein
MANOVA	Multivariate Analysis of Variance
MetS	Metabolic Syndrome
MHR	Maximum Heart Rate

MS	Metabolic Syndrome Study Group
NCEP	National Cholesterol Education Panel
PRO	Protein
REE	Resting Energy Expenditure
SBP	Systolic Blood Pressure
T2DM	Type 2 Diabetes Mellitus
TC	Total Cholesterol
TG	Triglycerides
TLC	Therapeutic Lifestyle Change
VO ₂	Volume of Oxygen Consumption
WC	Waist Circumference
WHO	World Health Organization

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CHAPTER I

INTRODUCTION

BACKGROUND

Metabolic Syndrome is a combination of health markers that has a direct effect on atherosclerotic disease. While there are multiple definitions, the general trend embraces risk factors such as abdominal obesity, insulin resistance, atherogenic dyslipidemia, elevated blood pressure, and elevated plasma glucose [1]. These markers have an additive effect on disease risk, increasing the risk for cardiovascular disease two-to-four times and elevating the risk of type 2 diabetes five-to-nine times [2-7]. The National Cholesterol Education Program – Adult Treatment Panel III created a definition for metabolic syndrome in 2001 that has been widely accepted and validated [2, 8-11]. The modified ATP III criteria, published in 2004 states that for a woman to be diagnosed with metabolic syndrome, she must have at least three of the following: central obesity (waist circumference >88 cm), hypertriglyceridemia (fasting triglycerides ≥ 1.7 mmol/L), low levels of high density lipoprotein cholesterol (<1.3 mmol/L), hypertension (blood pressure $\geq 135/85$ mmHg or taking medication), and/or fasting plasma glucose ≥ 5.6 mmol/L) [8, 12].

In the United States, it is estimated that one-quarter of the adult population has metabolic syndrome [13], which was predicted to include 47 million Americans in 2000 [14], and may be as high as 50-75 million now [15]. Obesity may be to blame for this rise, as prevalence of metabolic syndrome increases in a graded fashion with body mass index [16, 17]. Obesity affects over 35% of US adults [18], and approximately one-third

of individuals who are overweight or obese manifest the metabolic syndrome condition [1, 17, 19, 20]. This becomes a larger concern for women, as two million more women than men are categorized as obese [21, 22] and the leading cause of death for women is cardiovascular disease [23, 24]. Unfortunately, it does not appear that this prevalence will diminish any time soon, as younger generations are already experiencing these health deficits. Nearly one million children in the US express the metabolic syndrome condition [25, 26], and more than 17% of adolescents are obese [27].

Most individuals with metabolic syndrome could benefit greatly from therapeutic lifestyle modification [3, 8, 17]. A combination of proper nutrition with physical activity is likely the best prescription for preventing and reversing the markers of metabolic syndrome. However, regarding the treatment of this condition, the most beneficial nutrition protocol is still heavily debated.

The National Cholesterol Education Program – Adult Treatment Panel III promotes a ratio of 15% protein, 50-60% carbohydrate, and 25-30% of caloric intake from fat for individuals with metabolic syndrome [17]. However, many other recommendations on the specific proportions of protein, carbohydrate, and fat to consume are touted by various researchers and organizations [28-30]. A review of current research suggests that reducing carbohydrate consumption can positively benefit triglyceride levels [31-40]. While there can be health benefits achieved by substituting some carbohydrate with either fat or protein, the addition of protein especially during a weight loss and exercise program will better preserve fat free mass and promote more optimal changes in body composition [41-43]. However that is not to say that higher

carbohydrate diets do not possess some health benefits; it may be that dietary guidelines should be individualized based on the patient's specific metabolic phenotype [43].

A dose-response relationship exists between low physical activity levels and increased cardiovascular disease risk and the individual components of metabolic syndrome [5, 44, 45]. Only 11-46% of the US adult population engages in the recommended amount of physical activity [46], and women are not as likely to reach the recommendations as men [47]. Therapeutic lifestyle change should focus on reversing the sedentary lifestyle, the atherogenic diet, and the overweight / obese status [33]. This is most effectively achieved with physical activity accompanied by weight reduction in order to target each element of metabolic syndrome individually [1, 7, 17, 48].

Moderate physical activity can improve the risk factors and/or reduce the likelihood of developing metabolic syndrome [8, 49]. Cardiorespiratory fitness is inversely correlated with the metabolic abnormalities of metabolic syndrome, cardiovascular disease, and type 2 diabetes mellitus. This is found to be true regardless of age, gender, ethnicity, or body weight [3, 6, 14, 24, 29, 50-53], and these health benefits have been noted at various durations and intensities [51, 53, 54]. Resistance exercise appears to produce preventative benefits as well, due to gains in muscle mass and strength, reduction in body fat, and improvement of insulin sensitivity [21, 42, 47].

Weight loss appears to be an integral component in the treatment of metabolic syndrome and corresponding diseases [8, 33, 55, 56]. A linear relationship exists between weight and waist circumference, blood pressure, lipid levels, and fasting glucose [50, 57, 58]. A decrease of seven-to-ten percent body weight over six-twelve

months is advised for patients with metabolic syndrome [27, 58-60]. Additional benefits may be obtained by placing focus on the loss of fat mass specifically [55].

A combination of dietary intervention with physical activity will best achieve the desired weight loss and reversal of metabolic syndrome risk factors. Specifically for women, a program that offers both higher carbohydrate and higher protein dietary options and also includes a 30-minute circuit-style resistance-training protocol three days a week may prove advantageous. Previous research from the Exercise and Sport Nutrition Lab has shown great success with weight and fat loss, maintenance or gain of fat free mass, maintenance of resting energy expenditure, and improvements in markers of fitness with this combined diet and exercise intervention [61, 62]. More recent research from the Exercise and Sport Nutrition Lab suggests that the higher protein diet may provide a more beneficial effect on insulin sensitivity as well as the aforementioned variables [63].

STATEMENT OF THE PROBLEM

When combined with an exercise and weight loss program, does a higher protein hypoenergetic diet promote a reduction in the prevalence of metabolic syndrome and each of the individual risk factors better than a higher carbohydrate hypoenergetic diet in overweight and sedentary women between the ages of 18-75?

PURPOSE

The purpose of this analysis was to determine whether following the higher protein diet intervention for ten-weeks promotes a reduction of metabolic syndrome and the individual NCEP ATP III metabolic syndrome risk factors better than the higher

carbohydrate diet for the same time period, when both diets are combined with a circuit-style resistance-based exercise program.

GENERAL STUDY OVERVIEW

This study is a retrospective analysis of eight ten-week weight loss studies that evaluated the effects of adherence to two dietary interventions while participating in a circuit-training based exercise program in women aged 18-75 years old. Female applicants who met the physician-approved criteria completed the ten-week diet and exercise protocol. Based on their responses on a carbohydrate tolerance questionnaire, participants were assigned to a hypocaloric diet either higher in protein or in carbohydrate and partook in a circuit-based resistance-training program. After study completion, participants were retrospectively categorized based on their baseline risk level for metabolic syndrome (apparently healthy versus metabolic syndrome; $<$ or \geq three ATP III risk factors respectively) to determine whether the macronutrient content of the diet combined with the exercise program differentially effected women designated as having metabolic syndrome. Additionally, further analysis was performed categorizing participants based on each metabolic syndrome risk factor independently (low versus high waist circumference, low versus high triglycerides, high versus low HDL cholesterol, low versus high blood pressure, and low versus high fasting glucose), in order to ascertain how the different dietary protocols affect each risk factor individually.

Dietary intake, anthropometric measurements, resting energy expenditure, body composition, and serum clinical chemistry samples were assessed at 0 and 10 weeks.

Participants also performed a maximal cardiopulmonary exercise stress test as well as lower and upper body muscular strength and endurance tests during each assessment. Questionnaires related to quality of life and body image were also completed at each session.

Hypotheses

Diet

H₁: There will be a statistically significant difference in macronutrient intakes between the two diet groups.

Metabolic Syndrome

H₂: There will be statistically significant differences in the markers of metabolic syndrome in participants with and without the presence of three or more metabolic syndrome risk factors.

Metabolic Syndrome Risk Factor Group

H₃: There will be statistically significant differences observed in changes in body composition as a result of diet intervention and/or the presence of three or more metabolic syndrome risk factors.

H₄: There will be no statistically significant differences observed in resting energy expenditure as a result of diet intervention and/or the presence of three or more metabolic syndrome risk factors.

H₅: There will be no statistically significant differences observed in resting hemodynamic parameters as a result of diet intervention and/or the presence of three or more metabolic syndrome risk factors.

H₆: There will be statistically significant differences observed in blood lipids as a result of diet intervention and/or the presence of three or more metabolic syndrome risk factors.

H₇: There will be statistically significant differences observed in markers of glucose homeostasis as a result of diet intervention and/or the presence of three or more metabolic syndrome risk factors.

H₈: There will be no statistically significant differences observed in fitness parameters as a result of diet intervention and/or the presence of three or more metabolic syndrome risk factors.

H₉: There will be no statistically significant differences observed in psychometric parameters (as measured via questionnaires regarding quality of life and body image) as a result of diet intervention and/or the presence of three or more metabolic syndrome risk factors.

Waist Circumference Metabolic Syndrome Risk Factor

H₁₀: There will be statistically significant differences observed in changes in body composition as a result of diet intervention and/or the presence of the waist circumference metabolic syndrome risk factor.

H₁₁: There will be no statistically significant differences observed in resting energy expenditure as a result of diet intervention and/or the presence of the waist circumference metabolic syndrome risk factor.

H₁₂: There will be no statistically significant differences observed in resting hemodynamic parameters as a result of diet intervention and/or the presence of the waist circumference metabolic syndrome risk factor.

H₁₃: There will be statistically significant differences observed in blood lipids as a result of diet intervention.

H₁₄: There will be no statistically significant differences observed in blood lipids as a result of the presence of the waist circumference metabolic syndrome risk factor.

H₁₅: There will be statistically significant differences observed in markers of glucose homeostasis as a result of diet intervention and/or the presence of the waist circumference metabolic syndrome risk factor.

H₁₆: There will be no statistically significant differences observed in fitness parameters as a result of diet intervention and/or the presence of the waist circumference metabolic syndrome risk factor.

H₁₇: There will be no statistically significant differences observed in psychometric parameters (as measured via questionnaires regarding quality of life and body image) as a result of diet intervention and/or the presence of the waist circumference metabolic syndrome risk factor.

Triglyceride Metabolic Syndrome Risk Factor

H₁₈: There will be statistically significant differences observed in body composition as a result of diet intervention.

H₁₉: There will be no statistically significant differences observed in body composition as a result of the presence of the triglyceride metabolic syndrome risk factor.

H₂₀: There will be no statistically significant differences observed in resting energy expenditure parameters as a result of diet intervention and/or the presence of the triglyceride metabolic syndrome risk factor.

H₂₁: There will be no statistically significant differences observed in resting hemodynamic parameters as a result of diet intervention and/or the presence of the triglyceride metabolic syndrome risk factor.

H₂₂: There will be statistically significant differences observed in blood lipids as a result of diet intervention and/or the presence of the triglyceride metabolic syndrome risk factor.

H₂₃: There will be statistically significant differences observed in markers of glucose homeostasis as a result of diet intervention and/or the presence of the triglyceride metabolic syndrome risk factor.

H₂₄: There will be no statistically significant differences observed in fitness parameters as a result of diet intervention and/or the presence of the triglyceride metabolic syndrome risk factor.

H₂₅: There will be no statistically significant differences observed in psychometric parameters (as measured via questionnaires regarding quality of life and body image) as

a result of diet intervention and/or the presence of the triglyceride metabolic syndrome risk factor.

HDL Cholesterol Metabolic Syndrome Risk Factor

H₂₆: There will be statistically significant differences observed in body composition as a result of diet intervention.

H₂₇: There will be no statistically significant differences observed in body composition as a result of the presence of the HDL metabolic syndrome risk factor.

H₂₈: There will be no statistically significant differences observed in resting energy expenditure parameters as a result of diet intervention and/or the presence of the HDL metabolic syndrome risk factor.

H₂₉: There will be no statistically significant differences observed in resting hemodynamic parameters as a result of diet intervention and/or the presence of the HDL metabolic syndrome risk factor.

H₃₀: There will be statistically significant differences observed in blood lipids as a result of diet intervention and/or the presence of the HDL metabolic syndrome risk factor.

H₃₁: There will be statistically significant differences observed in markers of glucose homeostasis as a result of diet intervention.

H₃₂: There will be no statistically significant differences observed in markers of glucose homeostasis as a result of the presence of the HDL metabolic syndrome risk factor.

H₃₃: There will be no statistically significant differences observed in fitness parameters as a result of diet intervention and/or the presence of the HDL metabolic syndrome risk factor.

H₃₄: There will be no statistically significant differences observed in psychometric parameters (as measured via questionnaires regarding quality of life and body image) as a result of diet intervention and/or the presence of the HDL metabolic syndrome risk factor.

Blood Pressure Metabolic Syndrome Risk Factor

H₃₅: There will be statistically significant differences observed in body composition as a result of diet intervention.

H₃₆: There will be no statistically significant differences observed in body composition as a result of the presence of the blood pressure metabolic syndrome risk factor.

H₃₇: There will be no statistically significant differences in resting energy expenditure as a result of diet intervention and/or the presence of the blood pressure metabolic syndrome risk factor.

H₃₈: There will be no statistically significant differences observed in resting hemodynamic parameters as a result of diet intervention.

H₃₉: There will be statistically significant differences observed in resting hemodynamic parameters as a result of the presence of the blood pressure metabolic syndrome risk factor.

H₄₀: There will be statistically significant differences observed in blood lipids as a result of diet intervention.

H₄₁: There will be no statistically significant differences observed in blood lipids as a result of the presence of the blood pressure metabolic syndrome risk factor.

H₄₂: There will be statistically significant differences observed in markers of glucose homeostasis as a result of diet intervention.

H₄₃: There will be no statistically significant differences observed in markers of glucose homeostasis as a result of the presence of the blood pressure metabolic syndrome risk factor.

H₄₄: There will be no statistically significant differences observed in fitness parameters as a result of diet intervention and/or the presence of the blood pressure metabolic syndrome risk factor.

H₄₅: There will be no statistically significant differences observed in psychometric parameters (as measured via questionnaires regarding quality of life and body image) as a result of diet intervention and/or the presence of the blood pressure metabolic syndrome risk factor.

Glucose Metabolic Syndrome Risk Factor

H₄₆: There will be statistically significant differences observed in body composition as a result of diet intervention.

H₄₇: There will be no statistically significant differences observed in body composition as a result of the presence of the glucose metabolic syndrome risk factor.

H₄₈: There will be no statistically significant differences observed in resting energy expenditure parameters as a result of diet intervention and/or the presence of the glucose metabolic syndrome risk factor.

H₄₉: There will be no statistically significant differences observed in resting hemodynamic parameters as a result of diet intervention and/or the presence of the glucose metabolic syndrome risk factor.

H₅₀: There will be statistically significant differences observed in blood lipids as a result of diet intervention and/or the presence of the glucose metabolic syndrome risk factor.

H₅₁: There will be statistically significant differences observed in markers of glucose homeostasis as a result of diet intervention and/or the presence of the glucose metabolic syndrome risk factor.

H₅₂: There will be no statistically significant differences observed in fitness parameters as a result of diet intervention and/or the presence of the glucose metabolic syndrome risk factor.

H₅₃: There will be no statistically significant differences observed in psychometric parameters (as measured via questionnaires regarding quality of life and body image) as a result of diet intervention and/or the presence of the glucose metabolic syndrome risk factor.

Delimitations

The parameters of this study were as follows:

1. Sedentary, overweight women (body mass index $>27 \text{ kg/m}^2$) between the ages of 18-75 were recruited for the studies in this analysis.
2. Recruitment was based on flyers distributed at physicians' offices, in local newspapers and television channels, on the Internet, and through campus mail.

3. Familiarization sessions as well as all testing was performed in the Exercise and Sport Nutrition Lab at Baylor University or Texas A&M University.
4. Assignment to the higher protein or higher carbohydrate diet groups was based on participant response on a Carbohydrate Tolerance Questionnaire.
5. Subjects had been sedentary for at least three months prior to the start of the study.
6. Participants had not utilized nutritional supplements that would affect muscle mass, anabolic/catabolic hormone levels, or weight loss for at least three months prior to the start of each study.
7. Subjects were required to obtain consent from their physician if they had any diagnosed controlled metabolic disorders.

Limitations

1. Recruitment was limited to Baylor University, Texas A&M University and the surrounding Waco and College Station communities, specifically to those who responded to the advertisements. Due to the non-random nature, this may affect the application on the general population.
2. Participation was limited to those who were self-motivated to respond to the advertisement, and additional incentives were given for completing the program. This may also affect the application on the general population.
3. Participants were required to follow the assigned nutrition plan in addition to completing the exercise circuit three times a week.
4. Participants were required to follow the nutrition and exercise program within a free-living environment.

5. The exercise protocol utilized (Curves®) has an associated cost, which may limit application to those who can afford to participate.
6. Participant information regarding tobacco use was not available within the database. Cigarette smoking is a large risk factor for cardiovascular disease and could also affect many of the metabolic syndrome variables.
7. Ethnicity data was not available in the database utilized. The prevalence (and potentially the causes) of metabolic syndrome varies by ethnicity, and analysis related to this topic was unable to be performed due to unavailable information. However, the population was a diverse representation of Central Texas including Caucasian, Hispanic, and African American individuals.
8. The NCEP ATP III definition of metabolic syndrome is not specific as to whether the blood pressure indicator is systolic ≥ 130 mmHg and diastolic ≥ 85 or whether it is either/or. For this study, we identified individuals with either elevated systolic or diastolic as having the blood pressure risk factor for metabolic syndrome.
9. Inherent limitations exist in the laboratory equipment utilized for collection and analysis of the data.

Assumptions

1. Participants were honest when answering the screening questions for entrance into the study, as well as questionnaires and food logs throughout the study.
2. Participants followed the assigned dietary protocol as specified.
3. Participants fasted for 12 hours prior to each testing session.
4. Participants abstained from exercise for 24 hours prior to each testing session.

5. Participants exerted maximal effort on the maximal treadmill and strength tests.
6. Participants notified the staff in the case of any adverse events.
7. Laboratory equipment was properly calibrated and functional for all testing sessions.
8. The population sample was normally distributed.
9. There was equal variability between the groups.

CHAPTER II

REVIEW OF THE LITERATURE

INTRODUCTION

As a health concern for close to a century, metabolic syndrome (MetS) has carried numerous aliases to include the deadly quartet, plurimetabolic syndrome, insulin resistance syndrome, dysmetabolic syndrome, and syndrome X [14, 51, 64]. While several organizations have unique criteria for MetS, the general trend includes risk factors that have a direct effect on atherosclerotic disease, including abdominal obesity, insulin resistance, atherogenic dyslipidemia, elevated blood pressure, and elevated plasma glucose [1]. Some individuals argue that MetS is imprecisely defined, and doubt the value of MetS as a cardiovascular disease (CVD) risk factor and its designation as a syndrome [48]. However, various definitions and numerous combinations in the expression of risk factors truly portray a “syndrome,” in which the risk factor clustering occurs in greater likelihood than could be expected by chance [1, 7, 65]. This is not just a “disease” or a discrete entity with a uniform pathology or treatment [1, 7].

A timely diagnosis of metabolic syndrome is critical, as early intervention may help prevent the future development of type 2 diabetes mellitus (T2DM), CVD, and other chronic diseases [48, 66, 67]. The individual risk factors of MetS have an additive effect on disease risk, leading to an increase in risk for CVD by two-to-four fold and elevate the risk of T2DM by as much as five-to-nine fold [2-7]. Additionally, a diagnosis of T2DM along with MetS increases the CVD risk more than either condition independently [17, 68].

Three of the more prevalent definitions of MetS are outlined by the World Health Organization, the National Cholesterol Education Program – Adult Treatment Panel III (NCEP ATP III), and the International Diabetes Foundation, and are defined in Table 2.1. Each definition embraces precise criteria that must be present in specified combinations of risk factors in order to create the metabolic syndrome profile for that organization.

Table 2.1: Criteria for the Diagnosis of Metabolic Syndrome in Women, by Organization

Clinical Measure	WHO (1998)	ATP III (2004 modified)	IDF (2005)
Insulin Resistance	IGT, IFG, or T2DM plus any 2 of the following:	N/A any 3 of the following:	N/A
Body Weight	WHR >0.85 or BMI >30 kg/m ²	WC ≥88 cm	WC ≥80 cm plus any 2 of the following:
Lipid	TG ≥1.7 mmol/L and/or HDL <1.0 mmol/L	TG ≥1.7 mmol/L and/or HDL <1.3 mmol/L	TG ≥1.7 mmol/L† HDL 1.3 mmol/L†
Blood Pressure	≥140/90 mm Hg	≥135/85 mm Hg†	≥130 mm Hg systolic ≥85 mm Hg diastolic†
Glucose	IGT, IFG, or T2DM	≥5.6 mmol/L (includes diabetes)	≥5.6 mmol/L (includes diabetes)
Other	Microalbuminuria		
<p>Table adapted from Diagnosis and Management of the Metabolic Syndrome, Grundy et al [1]. Values listed are specific to women. BMI, body mass index; HDL-C, high density lipoprotein cholesterol; IFG, impaired fasting glucose; IGT indicates impaired glucose tolerance; T2DM, type 2 diabetes mellitus; WHR, waist-to-hip ratio; TG, triglycerides. †Criteria includes a Rx for that clinical measure.</p>			

For the World Health Organization (WHO) definition (identified in 1998), insulin resistance (or glucose intolerance or diagnosed diabetes) is a requirement for metabolic syndrome, plus the addition of at least two other risk factors: obesity (body mass index (BMI) >30 kg/m² or waist-to-hip ratio >0.85), dyslipidemia (fasting triglycerides ≥1.7

mmol/L or HDL cholesterol <1.0 mmol/L), hypertension (blood pressure >140/90 mmHg), and/or microalbuminuria (albumin excretion >20 µg/min) [8]. The values listed in this report focus specifically on diagnosis for women.

The NCEP ATP III identifies six components of MetS that relate to CVD; abdominal obesity, atherogenic dyslipidemia (to include elevated triglycerides (TG), small low-density lipoprotein (LDL) particles, and/or low levels of high-density lipoprotein (HDL) cholesterol), elevated blood pressure (BP), insulin resistance (with or without glucose intolerance), a proinflammatory state, and a prothrombotic state [14, 19]. While the NCEP ATP III definition (created in 2001 and modified in 2004) does not embrace any particular risk factor as a mandatory prerequisite, a female must have at least three of the following to meet their diagnosis of MetS: central obesity (waist circumference (WC) >88 cm), hypertriglyceridemia (TG ≥1.7 mmol/L), low HDL cholesterol (<1.3 mmol/L), hypertension (BP ≥135/85 mmHg or taking medication), and/or fasting plasma glucose (≥5.6 mmol/L or diagnosed diabetes) [8]. This “grab bag” definition, as it has been described, allows for ten possible phenotypes of MetS, nine of which include a lipid abnormality [3, 4].

The International Diabetes Foundation (IDF) offers a newer definition (created in 2005) in which central obesity is a mandatory prerequisite (WC ≥80 cm for women) and two additional factors are required for diagnosis: glucose >5.6 mmol/L (or diagnosed diabetes), HDL cholesterol <1.29 mmol/L (or drug treatment for low HDL), TG ≥ 1.7 mmol/L (or drug treatment for high TG), and/or BP ≥ 130/85 mmHg (elevated systolic and/or diastolic blood pressure, or drug treatment for hypertension) [3, 7]. IDF and

NCEP ATP III differ in the lower waist circumference and blood pressure criteria in IDF, which explains why previous statements claim that if the IDF definition were utilized more frequently, it would substantially increase the prevalence of MetS [17, 69, 70]. Yet, the NCEP ATP III criterion appears to be utilized most commonly.

In addition to abdominal obesity and insulin resistance, a variety of other conditions may influence MetS to include physical inactivity, aging, hormone imbalance, inflammation, impaired endothelial function, increased sympathetic nervous system activity, genetic abnormalities, fetal malnutrition, and environmental factors [1, 14, 20, 51, 71]. Furthermore, MetS is associated with numerous ailments including polycystic ovarian syndrome, non-alcoholic fatty liver disease, gallstones, sleep disturbances, sexual impotence, and some forms of cancer [15, 17]. In some cases, MetS may be the result of commonly prescribed medications such as corticosteroids, antidepressants, antihistamines and antipsychotics, likely due to their side effect of weight gain [17, 60, 72, 73].

Previous comparative research studies have considered whether a diagnosis of MetS is more beneficial than a diagnosis of each risk factor independently [48, 71, 74], and also whether one definition has more predictive capabilities over another [2, 8-11]. Kahn [48] has found the MetS definitions to be imprecise, and raises doubt in the predictive power of a metabolic syndrome diagnosis in forecasting diabetes. He further describes MetS as merely a multivariate risk factor for CVD and states that based on current definitions, MetS does not warrant designation as a syndrome. Stern et al [71, 74] and others [71, 74], also believe that independent of insulin resistance (and treatment

of diabetes if necessary), MetS may not have additional predictive capabilities for CVD risk. Yet many other researchers have found a diagnosis of MetS (using various organization's definitions) to be beneficial in predicting future disease risk. A comparison between the WHO and the NCEP ATP III definitions found that while the two definitions identified similar proportions of the population with MetS (25.1% and 23.9% respectively), the actual individuals that were identified varied by as much as 15-20% depending on the definition utilized (mainly by differences found within ethnicities), although both criteria were similar in their prediction of CVD risk [8, 9]. Another review of three analyses found the NCEP ATP III definition to be a slightly better predictor of CVD and all-cause mortality in two out of the three studies evaluated [2, 10, 11]. It has been stated that while the WHO definition may be the most useful for research purposes, the NCEP ATP III may be more applicable for clinical practice [8].

PREVALENCE

Metabolic syndrome is quite common, and prevalence increases with both age and BMI. In the United States alone, approximately one-quarter of the adult population (~24% of men and ~23.4% of women) is diagnosed with MetS [13, 20]. In the year 2000, 47 million Americans had MetS [14], and this number was projected to reach 50-75 million by 2010 [15]. While only ~6.7% of twenty-year-olds have MetS, greater than 40% of individuals over 60 may have this disease [14, 17, 19]. The third report by the National Health and Nutrition Examination Survey found that MetS increases with BMI in a graded fashion; where only 1-3% of individuals with a BMI between 18.5-20.9 kg/m², but 9.6-22.5% of individuals with a BMI of 25-26.9 kg/m² have the syndrome

[16, 17]. Likewise, data from this survey has also found that as BMI increases, the prevalence for each individual risk factor also increases [19].

Obesity

Obesity is a major risk factor for MetS and may be to blame for the rise in the syndrome's prevalence. Approximately one-third of individuals who are overweight or obese manifest the MetS condition [1, 17, 19, 20]. Due to this potential correlation, it is important to note that obesity affects more than one-third (35.7%) of US adults [18]. Additionally, obesity prevalence is on the rise. Two separate analyses have been performed to predict the prevalence of obesity in the US by 2030. Finkelstein et al [18] utilized a non-linear trend and data from the 1990-2008 Behavioral Risk Factor Surveillance System to predict obesity rising as high as 42% of the population. Meanwhile, Wang et al [18, 75] used a linear trend with data from the 1970's-2004 National Health and Nutrition Examination Survey reports, and estimate obesity affecting 51% of the US population by 2030! This continual rise in obesity, whatever the estimated percentage, causes concern for a continued increase in the prevalence of MetS as well.

Global and Adolescence

Metabolic syndrome and the related illnesses are not only a concern in the US, nor exclusive to adults. On a global scale, WHO reported that 60% (35 of 58 million) of deaths in 2005 were a result of chronic diseases such as CVD and T2DM, and predict a 17% increase over the next decade [76, 77]. Additionally, obesity is estimated to affect 312 million people worldwide [13].

In regards to adolescents, it is suspected that nearly one million children have MetS in the US alone [25, 26]. Obesity affects over 17% of US children and adolescents [27], and the number of children who are overweight or obese has doubled over the last few decades [78]. This suggests that the prevalence of childhood MetS is also likely to rise. Lawson [79] has estimated that greater than four million US adolescents and young adults have a risk level for MetS equivalent to individuals greater than 60 years old, based on their level of obesity.

CONCERNS FOR WOMEN

There are also specific concerns regarding women and the metabolic syndrome. While the prevalence of MetS is similar in both genders, women are reported as having a higher morbidity related to the disease [80, 81]. According to the 2003-2006 National Health and Nutrition Examination Survey report [19], abdominal obesity, hypertension, and hyperglycemia are the MetS risk factors of highest prevalence in women at 53%, 40%, and 39% respectively. This report additionally mentions that the prevalence of many of the risk factors (abdominal obesity, hypertriglyceridemia, hypertension, and hyperglycemia) increase with each successive age bracket, and also found overweight and obese females to be five and 17 times as likely, respectively, to meet the criteria for MetS as normal weight females. In the US, two million more women than men are categorized as obese [21, 22], the leading cause of death for women is CVD [23, 24], and annual CVD mortality exceeds the mortality rate from breast cancer in women younger than 50 [81, 82]. These health issues may be either the cause or effect of MetS.

Two additional concerns for women are heredity and menopause. A comparison of National Health and Nutrition Examination Survey data between 1988-2004 states that daughters of mothers diagnosed with diabetes were almost twice as likely as other women to either express the MetS phenotype (adjusted odds ratio 1.96), be obese (adjusted odds ratio 1.7-2.1), have elevated plasma glucose levels (adjusted odds ratio 1.9), and/or have low levels of HDL (adjusted odds ratio 1.6) [81]. Additionally, menopause relates to an increase in abdominal obesity, a shift to a more atherogenic lipid profile (higher total cholesterol (TC), LDL, and TG, and lower HDL), and a four-fold increase in CVD risk [50]. This data suggests that the high prevalence of MetS and related illnesses found today will have a detrimental impact on future generations of women as well.

PREVIOUS RESEARCH ON THE TREATMENT OF METABOLIC SYNDROME

Individuals with high short-term risk for CVD may need drug therapy for distinct MetS risk factors, however most individuals with MetS could benefit greatly from therapeutic lifestyle modification (TLC) [3, 8, 17]. While the primary focus for reducing CVD risk is LDL cholesterol reduction (which is not a MetS risk factor) and smoking cessation, the treatment of MetS and accompanying risk factors is the secondary aim [1]. Modification of hypertension and dyslipidemia can decrease the risk of CVD, while glucose regulation can reduce the risk of T2DM [51]. TLC should focus on reversing the sedentary lifestyle, the atherogenic diet, and the overweight / obese status [33]. This is most effectively achieved with physical activity accompanied by weight reduction (a

reasonable goal is ~10% in one year), which will aid insulin resistance modification and target each element of MetS individually [1, 7, 17, 48]. Even when weight loss is not achieved, physical activity may be beneficial in decreasing abdominal fat and improving insulin sensitivity [51]. Furthermore, research from the Exercise and Sport Nutrition Lab (ESNL) [62, 83], as well as others [51], proves for exercise to effectively reverse MetS variables, it should be combined with appropriate dietary modification. Likewise, adding exercise to a nutritional intervention may be more successful than dieting alone [52]. The specific effects of various dietary modifications with and without physical activity are discussed in further detail below.

Nutrition

Until Ornish's published findings in 1998 [26, 84], stating that proper nutrition may be related to disease reversal and prevention, clinicians thought heart disease could only be reversed through surgery. In regards to nutrition advice for the treatment or prevention of MetS, there is agreement among clinicians and researchers concerning general health guidelines, but disagreement on the specific dietary treatment. The consensus includes a recommendation for reduced consumption of simple sugars with an increased intake of fruits, vegetables, complex carbohydrates, whole grains and fiber [1, 7, 17, 21, 60, 85], a low intake of saturated fat, trans-fat, and cholesterol [1, 7, 17, 21, 33, 60], and the inclusion of fish, nuts, and low-fat dairy products [1, 17, 85]. Many also recommend reducing sodium intake specifically for hypertensive individuals [1, 7, 17]. The NCEP ATP III specifically promotes caloric balance (for weight maintenance) and a ratio of 15% protein (PRO), 50-60% carbohydrate (CHO), and 25-30% of caloric intake

from fat for individuals with MetS [17]. Additionally the NCEP ATP III recommends consuming 20-30 g/day of fiber, less than 200 mg/day of cholesterol, less than 7% kcal/day from saturated fat, and fat consumption made up of 10% polyunsaturated fat and 20% monounsaturated fat [17].

Specific dietary recommendations may be confusing to MetS patients, as all ends of the spectrum have been advised. Numerous research studies have attempted to define the most beneficial macronutrient composition to regulate/prevent MetS and/or the individual health components. For this review, a search was performed through EBSCO and Google Scholar, using queries with combinations of the following words or phrases: “metabolic syndrome” + “diet” + “protein” + “carbohydrate” + “weight” + “blood pressure” + “lipids” + “obesity”. The search took place between February 1-25, 2013 and excluded studies prior to the year 2000 as well as studies that did not report at least three of the risk factors for MetS. The review is divided into four categories: 1) research on carbohydrate restriction alone, 2) the adjustment of carbohydrate and fat consumption percentages, 3) the elevation of protein consumption, and 4) elevated protein consumption with specific exercise prescriptions.

Carbohydrate Restriction

There has been inconsistency in study findings regarding the relationship between MetS and carbohydrate (CHO) consumption [29]. Many researchers suggest that a high intake of CHO should be avoided [60], since high CHO diets (>60%) may elevate glucose, insulin, and TG levels [21, 29, 86, 87], and lower CHO consumption can more greatly decrease weight and TG levels and lead to an improvement in insulin

sensitivity [31-33]. Yet other studies have found an inverse correlation between high CHO consumption and prevalence of CVD [28, 88], or generally no significant relationship between CHO consumption and the prevalence of MetS and/or insulin resistance [29].

Table 2.2 identifies nine carbohydrate restriction studies with varying reductions in CHO allotment, from 20-70 grams a day. Seven of the nine studies found a significant decrease in TG [34-40], while the other two reported no significant difference [89, 90]. Of additional benefit to combatting MetS, all of the studies lead to a significant decrease in weight [34-39, 89, 90], five of the studies measured a reduction in blood pressure [34, 35, 39, 40, 89], two studies [35, 36] (and a third only in men [90]) found a significant increase in HDL, and one (severely low CHO) study also showed a significant decrease in fasting glucose [38] (with another study showing significance only in men [90]). Furthermore, many studies also measured a decrease in other health markers such as TC and LDL, two of the studies lead to a significant decrease in body fat percentage [34, 35], and one study specifically calculated a decrease in prevalence of MetS by 50% when consuming only 10-25% daily calories from CHO [89]. The benefits of reducing CHO consumption are evident, yet the question still remains as to which macronutrient most appropriately fills the necessary caloric requirement.

Table 2.2: Carbohydrate Restriction for Treatment of Metabolic Syndrome Risk Factors

Authors (Date)	Study Length Study Population	Groups	Dietary Protocol (%PRO:CHO:FAT)		Exercise	Wt	BP	TG	HDL	Glu	Other	
Meckling, Gauthier, Grubb, Sanford (2002) [34]	8 weeks	reduced carb and calorie	~70g/day CHO	~5740 kJ/day (deficit of ~2644 kJ/day)	maintain normal activity levels	-6.1%*	SBP - 7.5%* DBP -8%*	↓	NS	NS	TC ↓ LDL ↓ Body fat % ↓	
	20 women; ~34.4 y/o, BMI~30.7											
Westman et al (2002) [35]	6 months	very low carb	<25 g/d CHO until achieved 40% target weight loss; then 50 g/d	no limit on caloric intake	encouraged to exercise ≥20 min 3x/wk	-10.3%*	SBP↓ DBP↓	-43.1%*	+19.2% *	---	TC -5.1%* LDL -7.4%* body fat % ↓	
	41 overwt/ obese healthy (31 fem); ~43.7 y/o, BMI~31.4											
Hickey et al (2003) [36]	~30-40 weeks (retrospect follow-up)	carb restricted (CRD)	<20 g/day CHO	CHO gradually added back in once weight loss or risk reduction achieved.	---	-1.1%	---	-38%*	+13%*	---	TC -13%* LDL -16%*	
	80 patients with atherogenic dyslipidemia (27% women); ~66 y/o, BMI~28.1											
Boden et al (2005) [37]	2 weeks (inpatient) after normal diet for one week	low carb	only 21 g/d CHO no limits to fat and protein consumption		maintain normal activity levels	-1.8%	---	-35%*	NS	---	TC -10%* insulin sensitivity improved ~75%	
	10 obese (7 fem) T2DM; ~51 y/o, BMI~40.3											
Yancy et al (2005) [38]	16 weeks	low-carb ketogenic diet (LCKD)	<20 g/d CHO	unlimited meat, poultry, etc. Fats and oils not restrict.	encouraged to exercise aerobically 30 min 3x/wk	Wt -6.6%* WC -5.2%*	NS	-42%*	NS	-16.6%*	HbA1c -16%	
	21 overwt (1 fem) T2DM; ~56 y/o, BMI~42.2											

Table 2.2: Continued

Authors (Date)	Study Length Study Population	Groups	Dietary Protocol (%PRO:CHO:FAT)		Exercise	Wt	BP	TG	HDL	Glu	Other
Hayes et al (2006) [39]	12 weeks (2 phases; 2 and 10 weeks)	Phase I	28:10:62	similar to South Beach Diet	---	Wt -3.3% WC -1.2%	SBP -6.2% DBP -8.6%	-43.4%*	NS	NS	---
	20 overwt/obese (16 fem) MetS; ~47.5 y/o, BMI~33.9	Phase II	30:27:43			Wt -5.6%* WC -4.5%*		-16.5%			
Miller et al (2007) [89]	3 months (2 phases: 2 and 10-11 wks)	Phase 1 (2 weeks)	28:10:62	No specific caloric restrictions given	---	Wt -5.4%* WC -5.0%*	SBP - 5.9%* DBP - 9.0%*	NS	NS	NS	prevalence of MetS -50%
	21 adults (81% fem) MetS (ATP III); ~47 y/o, BMI~33.8	Phase 2 (10-11 weeks)	30:27:43								
Muzio et al (2007) [40]	5 months	low carb	19:48:33	~500 kcal/day defect from estimated expenditure	encouraged to increase PA	NS	↓	↓	NS	decrease	---
	100 (73 fem); ~52.4 y/o, BMI~37.2	high carb	13:65:22			NS	decrease	dec			LDL: ↓
Sasakabe, Haimoto, Umegaki, Wakai (2011) [90]	6 months	moderate low-carb diet (LCD)	HbA1c <9%, cut CHO from dinner; HbA1c ≥9%, cut CHO from bfast and dinner	no specific ratio prescribed; free consumption of PRO and FAT	maintain normal activity levels	Men -2.8% Women -3%	NS	NS	Men +10.6% * Women NS	Men -13.4%* Women NS	LDL: Women -15.3%* Men NS
	52 with T2DM (24 fem); ~60.0 y/o, BMI~24.5										
BMI, (body mass index) values are listed in kg/m ² . BP, blood pressure; CHO, carbohydrate; DBP, diastolic blood pressure; Fem, females; Glu, glucose; HbA1c, hemoglobin A1c; LDL, low density lipoprotein cholesterol; PA, physical activity; SBP, systolic blood pressure; TC, total cholesterol; TG, triglycerides; WC, waist circumference; Wt, weight; y/o, years old. Values are calculated percent change from baseline. Listed values are significantly different from baseline, representing a time effect. Bold and * indicate p<0.05 for group/diet effect. Within a study, values that do not share a common superscript are significantly different. ---, value not reported or measured; NS, no significant change from baseline; NDE, no diet effect; ⬆⬇ significant change, yet specific value not reported.											

Carbohydrate Versus Fat Consumption

Similar to carbohydrates, the recommended percentage of daily fat consumption also varies greatly. As previously mentioned, the NCEP ATP III suggests no more than 30% fat [17], yet others recommend a fat intake of 35% or even as high as 40% can be beneficial [1, 28]. While some argue that the percentage of fat intake is unrelated to the markers of MetS [29, 30], others contend that humans are largely sensitive to fat consumption and that there is a delicate window (25-35%) of fat consumption for optimal lipid level regulation [1]. Certainly the content of fat must be considered. Consumption of saturated fats has been proven to promote dyslipidemia and lead to atherogenesis [85], while unsaturated fatty acid consumption, specifically monounsaturated fat, can reduce the prevalence of CVD [28].

More than just considering each macronutrient individually, Grundy and colleagues [28] raise the question of whether the specific ratio of fat-to-CHO affects MetS risk factors. Interestingly, Brunner et al [29, 30] found no relationship between any of the macronutrients (PRO, CHO, or fat) to the prevalence of MetS. However, this review aims to dig deeper.

The literature search returned 24 studies on CHO vs. fat consumption that met the criteria for this review. Of these studies, two focus on the comparison of simple vs. complex carbohydrates [91, 92], one compares a low-fat diet group to a control [93], two compare popular dietary programs (such as Atkins and the American Diabetes Association) [94, 95], and 19 specifically measure the effects of various carbohydrate versus fat ratios [31, 96-113]. These 24 studies are summarized in Table 2.3.

Table 2.3: Carbohydrate versus Fat Restriction for Treatment of Metabolic Syndrome Risk Factors

Authors (Date)	Study Length Study Population	Groups	Dietary Protocol (%PRO:CHO:FAT)		Exercise	Wt	BP	TG	HDL	Glu	Other
Rodriguez-Villar et al (2000) [96]	6 weeks; crossover design (no wash out)	high-carb (CHO)	15:55:30	isocaloric diets, prescribed based on est. energy req.	maintain normal activity levels	NDE	---	NDE	NDE	NDE	---
	22 with T2DM; ~61 y/o, BMI~28.3	high MUFA (MUFA)	15:40:45 25% MUFA								
Noakes and Clifton (2000) [97]	12 weeks	very low fat (VLF)	19:71:10	~6500 kJ/day	maintain normal activity levels	Wt -9.7% WC -8%	↓ ^a	-8%	NS	---	TC -14.8% ^b LDL -19% ^b
	62 subjects; ~45.7 y/o, BMI~31.2	high sat fat (HSF)	18:50:32 17% from sat fat				↓ ^b	-23.1%			TC -8% ^a LDL -6.5% ^a
		high unsat fat (HUF)	20:48:32 6% from sat fat				↓ ^b	-21.9%			TC -17% ^b LDL -19.8% ^{a,b}
Saris et al (2000) [91]	6 months	control	dietary intervention typical of the average national intake		---	+0.9% ^a	---	NS	NS	NS	FM +1.8% ^a
	398 moderately obese (51% fem); ~39 y/o, BMI~30.4	high carb simple	low-fat food with simple:complex carb ratio of 1.5:0.5			-1% ^b					FM -3.8% ^b
		high carb complex	low-fat food with simple:complex carb ratio of 0.5:1.5			-2% ^b					FM -5.3% ^b
Poppitt et al (2001) [92]	6 months	control	maintain fat intake ~35-40%	encouraged to not reduce caloric intake	---	NS	NS	NS	↓	---	TC NS
	39 (27 fem) with MetS; ~46 y/o, BMI~32	low-fat, complex carb (LF-CC)	10% ↓ fat 1:2 carbs simple:complex			-4.7%*					TC -11.1%*
		low-fat, simple carb (LF-SC)	10% ↓ fat and consume 2:1 carbs simple:complex			NS					↑
Tuomilehto et al (2001) [93]	~3.2 years follow-up	control	general nutrition/exercise advice		---	Wt -0.9% WC -1.3%	SBP -0.7% DBP -3.5%	-0.6%	+2.1%	-0.9%	---
	522 overwt (350 fem) with IGT; ~55 y/o, BMI~31	low fat	fat consumption <30% <10% saturated fat, ≥15 g/1000 kcal fiber		moderate ex for 30 min/d	Wt - 4.7%* WC -4%*	SBP -3.6%* DBP -5.8%*	-11.7%*	+4.3% (p=.06)	-3.7%*	

Table 2.3: Continued

Authors (Date)	Study Length Study Population	Groups	Dietary Protocol (%PRO:CHO:FAT)		Exercise	Wt	BP	TG	HDL	Glu	Other
Landry et al (2003) [98]	7 weeks	high carb	13:60:27	ad lib	maintain normal activity levels	-2.9%	---	NS	---	-3.8%	---
	37 healthy males; ~34 y/o, BMI~28	low carb	13:46:41			-2%		-24%		NS	
Colette et al (2003) [99]	8 weeks	high CHO	20:55:25 10% from MUFA	30% ↓ initial energy intake	no exercise prescribed	-7.2%	---	NS	NS	NS	TC -7.5% LDL NS
	32 overwt (23 fem); ~49 y/o, BMI~35	high MUFA	20:40:40 25% from MUFA			-6.9%		-21.9%	-8.5%	-11.5%	TC -9.8% LDL -8.2%
Brehm, Seeley, Daniels, D'Alessio (2003) [100]	6 months	very low carb	20 g/day CHO for 2 weeks, increase to 40-60 g/day	ad libitum	maintain normal activity levels	-9.3%*	SBP -1.7% DBP -6.3%	-23.4%*	+13.4%	-9.1%	body fat - 12.8%*
	53 healthy, obese fem; ~44 y/o, BMI~33.6	low fat	15:55:30	-500 kcal/day baseline		-4.2%	SBP -1.7% DBP -1.3%	+1.6%	+8.4%	-4.0%	body fat -5.2%
Samaha et al (2003) [31]	6 month	low-carb	≤30 g CHO/day	no fat restrictions	---	-4.5%*	NS	-20%*	NS	all: -8.6%* T2DM: -15%*	---
	132 severely obese (23 fem); ~53.5 y/o, BMI ~43	low-fat	30% kcal/day from fat	-500 kcal/day deficit		-1.4%		-4%		all: -1.6% T2DM: -3.2%*	
Volek et al (2004) [101]	4 week period; cross-over design	very low carb	30:10:60	hypocaloric (-500 kcal/day)	maintain normal activity levels	---	---	-22.5%	+1.9%	-3.5%	TC:+1% LDL: +5.3% HOMA: - 14%*
	13 overwt/obese fem; ~34 y/o, BMI~29	low-fat	20:55:25					-11.2%	-7.7%*	+2.3% *	TC:-7%* LDL: -5.3%* HOMA: +27.3%
Miyashita et al (2004) [102]	4 weeks	low carb	25:40:35	low calorie: 1000 kcal/day	walking 2x/d for 30 min ea	-12.3%	---	-40-50%	↑	-49.8%	TC: -20%
	22 obese (6 fem) T2DM; ~52.4 y/o, BMI~27	high carb	25:65:10			-9.9%			NS	-49%	
Gerhard et al (2004) [103]	6 weeks; crossover (6-12 week wash)	low fat	15:65:20	10% energy refined sugar; diets 25%+ est energy req.	maintain normal activity levels	-1.5%*	---	NS	-7.1%	NS	---
	11 subjects (8 fem) T2DM; ~50.4 y/o, BMI~37.2	high- MUFA	15:45:40 26% MUFA			-0.5%		-4.5%			

Table 2.3: Continued

Authors (Date)	Study Length Study Population	Groups	Dietary Protocol (%PRO:CHO:FAT)		Exercise	Wt	BP	TG	HDL	Glu	Other
Meckling, O'Sullivan, Saari (2004) [104]	10 weeks	low-fat (LF)	<20% fat	-2540 kJ/day	maintain normal activity levels	-7.4%	SBP -9.2% DBP -6.4%	-25.4%	-15.4%	-10.2%	LBM: -1.7%*
	31 overwt/obese; ~42 y/o, BMI~32.2	low-carb (LC)	50-70 g/day of CHO	-3195 kJ/day		-9.9%	SBP -8.2% DBP -7.9%	-29.4%	+12.2%*	-8.0%	LBM: -3.3%
Yancy et al (2004) [105]	6 months	low-carb	20g/day CHO, gradually increased	high-pro, high-fat Atkins	encouraged to exercise 30 min 3x/wk	-12.9%*	NDE	-42%*	+9.8%*	---	TC: -3.3% LDL: +1%
	120 overwt, hyperlipidemic; ~44-45 y/o, BMI~34.5	low-fat	<30% fat, with <10% saturated fat	500-1000 kcal/day less than maint.		-6.7%		-14.4%	-2.9%		TC: -5.6% LDL: -5%
Shai et al (2008) [106]	2 years; 6 months wt loss 18 months maint	low-fat / restrict cal	<30% fat, <10% sat fat	1500 kcal/day women 1800 kcal/day men	---	-3.2% ^a	SBP -3.3% DBP -1.1%	-1.7%	+16.3%	NS	TC/HDL -12%
	322 moderately obese (14% fem); ~52 y/o, BMI 31	med / restrict cal	no more than 35% fat	cal, pro, and fat not limited		-4.8%^b	SBP -4.1% DBP -2.7%	NS	NS	-35%*	NS
		low-carb / no-restrict cal	20 g/day CHO for 2 months, increase to 120 g/day			-5.1%^b	SBP -3.0% DBP -1.0%	-13%*	+22.4%*	NS	TC/HDL - 20%*
Davis et al (2009) [107]	1 year	low-carb	20-25 g/day CHO, gradually increased	modified Atkins diet	rec 150 min/wk physical activity	-3.3%	SBP +1.6% DBP -4.0%	-10.7%	+12.3%*	---	---
	105 overwt adults with T2DM; ~54 y/o, BMI~36	low-fat	25% fat	modeled after DPP		-3.1%	SBP -6.0% DBP -2.9%	-0.7%	+5%		
Brehm et al (2009) [108]	1 year	high MUFA	15:55:30 20% MUFA	-200-300 kcal/day from calc expend.	encouraged to walk 30 min/d several d/wk	-3.9%	SBP -1.5% DBP -6.4%	NS	+11.9%	-5.3%	---
	124 overwt/obese (61 fem) T2DM, ~56.5 y/o, BMI ~35.9	high CHO	15:60:25			-3.7%	SBP -0.8% DBP -5.2%		+11.6%	-5.9%	
Iqbal et al (2009) [113]	24 months	low-carb	<30g/day CHO; no restrictions on fat/cal intake	weekly nutrition sessions for one month; monthly thereafter	encouraged to exercise 30 min 5x/wk	-1.3%	SBP -8.0% DBP -4.8%	-16.5%	+1.7%	-1.1%	---
	144 obese, diabetic (15 fem); ~60 y/o, BMI~37.5	low-fat	≤30% cal fat with ~500 kcal / day			-0.2%	SBP -3.2% DBP -5.4%	-7.8%	+2.0%	-3.0%	
Mueller et al (2010) [109]	140 days total; 70 days fed, 70 days own	carb- controlled	20:30:50	-500-700 kcal/d, adjusted from indirect calorimetry	---	-10%	---	-23.3%	-0.5%	-3.1%	---
	16 (15 fem, mostly non-white), ~46-49 v/o, BMI~39.5	fat- controlled	20:50:30			-8.2%		+1.3%	+2.8%	-3.8%	

Table 2.3: Continued

Authors (Date)	Study Length Study Population	Groups	Dietary Protocol (%PRO:CHO:FAT)		Exercise	Wt	BP	TG	HDL	Glu	Other
Foster et al (2010) [110]	2 years	low carb	20 g/day CHO for 3 months, gradually increased	No pro or fat limit	walking 4 x/week, progressing from 20-min to 50- min/session by 19 wks	-6.1%	SBP -2.2% DBP -2.6%	24 mo: - 10.8%	+16.8%*	---	LDL -4.0%
	307 subjects; ~45.5 y/o, BMI ~36.1	low-fat	15:55:30	1200-1800 kcal/day		-0.7%	SBP -2.1% DBP -0.7%	24 mo: - 11.8%	+10.22%		LDL -6.5%
Goldstein et al (2011) [94]	12 months; 1 month DASH, 3 months supervision, 8 months follow up	modified Atkins (ATK)	25 g/CHO daily increasing to 40g/day after 6 weeks	cal, pro, and fat not limited	advised aerobic activity for 30 min, 3 x/week	-3.7%	SBP -9.9% DBP -10.5%	-19.5%	+10%	+71.7%	---
	52 T2DM; ~56 y/o, BMI~33.2	ADA cal- restrict (ADA)	20:40-45:35-40	Men 1500 kcal/day women 1200 kcal/day		-5.9%	SBP -3.7% DBP -4.7%	-2.0%	+12.3*	+20.2%	
Rajaie, Azadbakht, Khazaei, Esmailzadeh (2012) [111]	6 weeks; crossover 2 week washout	high carb (HC)	20:60:20	Crossover between groups	maintain normal activity levels	WC -2.5%	SBP -2.4% DBP -2.2%	+0.1%	NS	NS	MetS prevalence: NS
	30 overwt/obese fem w MetS (ATP III); BMI>25	moderate restricted carb (MRC)	21:43:36			WC -3.7%	SBP -7.1% DBP -16%*	-18.1%			MetS prevalence: -35.8%*
Metkus, Dobrosielski, Stewart (2012) [112]	6 months	low carb	not specified	isocaloric	supervised exercise training	Wt: -13.3%* WC: 11.8%*	SBP -8% DBP -11.6%	-42%*	+15.3%*	-6.1%	---
	77 overwt/obese healthy; ~48.5 y/o	low fat	not specified			Wt: -8.3% WC: -5.9%		-6.3%	+1.5%		
Hussain et al (2012) [95]	24 week	Low cal diet (LCD)	reduced fat, lean meats	Diets were self-selected	---	D: Wt-7% WC-3.5% ND: Wt - 5.1% WC -2.8%	---	NS	NS	↓	TC NS LDL NS
	363 overwt/obese 76.3% fem (27% diabetic (D)); ~37.2 y/o, BMI~37.3	Low carb ketogenic diet (LCKD)	~20g/d CHO, gradually increasing 5g/d in later weeks			D: Wt -12%* WC-7.3%* ND: Wt - 12%* WC -6.1%*		↓	↑	↓*	TC ↓ LDL ↓
BMI (body mass index) values are listed in kg/m ² . BP, blood pressure; CHO, carbohydrate; DASH, dietary approaches to stop hypertension; DBP, diastolic blood pressure; DPP, diabetes prevention program; Fem, females; FM, fat mass; Glu, glucose; HDL, high density lipoprotein cholesterol; HOMA, homeostasis model assessment; IGT, impaired glucose tolerance; LBM, lean body mass; LDL, low density lipoprotein cholesterol; MetS, metabolic syndrome; MUFA, monounsaturated fatty acids; SBP, systolic blood pressure; T2DM, type 2 diabetes mellitus; TC, total cholesterol; TG, triglycerides; WC, waist circumference; Wt, weight; y/o, years old. Values are calculated percent change from baseline. Listed values are significantly different from baseline, representing a time effect. Bold and * indicate p<0.05 for group/diet effect. Within a study, values that do not share a common superscript are significantly different. ---, value not reported or measured; NS, no significant change from baseline; NDE, no diet effect; ⬆️⬆️ significant change, yet specific value not reported.											

Modifying the simple:complex CHO ratio without altering the fat ratio (or caloric intake) appears to have minimal effect on the markers of MetS and led to a decrease in HDL in all groups [92], while adding a low-fat diet component to the simple:complex CHO ratio also did not lead to an improvement in the markers of metabolic health [93]. Focusing on low fat consumption (<30% kcal/day) proved to significantly benefit almost all markers of MetS; weight, WC, BP, TG, and glucose, and led to a trend in improved HDL with a p-value of 0.06 [93]. In the diet comparisons, no statistically significant advantage was found between the Atkins (restricted carbohydrate) and American Diabetes Association (restricted calorie) diets for MetS markers [94], but Hussain et al [95] did find a significant improvement in all MetS markers measured (weight, WC, TG, HDL, and glucose) as well as TC and LDL for both diabetic and non-diabetic subjects using a low CHO ketogenic diet when compared to a low calorie reduced fat diet [95]. By keeping the PRO consumption ratio constant and altering the fat and CHO percentages, the lower CHO consumption did lead to reduced TG in seven of the 19 studies reviewed [31, 98-100, 105, 106, 112]. Additionally, eight of the studies reported a significant increase in HDL for the low-CHO (high-fat) group [99, 102, 104-107, 110, 112] (with an additional study reporting a significant decrease in HDL in the higher CHO group [101]), four lead to a significant decrease in weight [31, 100, 105, 112], two studies found a decrease in BP with lower CHO consumption [97, 111], and two showed a decrease in fasting glucose [31, 99] (with an additional study reporting a significant

increase in glucose in the higher CHO group [101]), while five studies found no significant differences between the diets [96, 103, 108, 109, 113] . These studies further promote CHO restriction for improving the markers of MetS, yet the lower fat diet also proved beneficial. This leads to the consideration of focusing on PRO consumption instead.

Increased Protein Consumption

A vast amount of research has proven higher protein (carbohydrate-restricted) intake to be beneficial for weight loss and markers of health [41, 42]. While the current recommended daily allowance of PRO is 0.8 g/kg/day [114, 115], most Americans consume more than 15% of their total energy intake from protein, which amounts to approximately 1.2-1.5 g/kg of body weight [115-118]. Greater quantities of PRO consumption have proven advantageous in retaining fat free mass (FFM) during weight loss [43], with higher quartiles of PRO intake resulting in greater FFM retention [42, 119]. Additionally, PRO intake >1.4g/kg/day may result in greater weight loss and body fat loss, as well as improved blood lipid profiles and glycemic control [43, 63, 120]. Placing focus on higher protein consumption (25-30% of energy, ≥ 1.2 g/kg/day) may be beneficial in the treatment of MetS based on the aforementioned factors as well as the reputation for protein to enhance satiety [43, 55], prevent a decrease in resting energy expenditure while dieting [62], improve muscle function and strength [118], and improve blood pressure [55].

Some researchers have expressed concern regarding potential health risks of high protein consumption (>2-3 times the RDA), in such that an increased acid load may lead

to increased calcium excretion with an increased osteoclast / decreased osteoblast activity and an overall negative calcium balance [115]. Meanwhile, the current recommended daily allowance has been based on epidemiological studies without an end goal of increasing muscle mass or function [118]. Elevated protein consumption, ≥ 2.0 g/kg/d has not been proven to be harmful [117, 118], and previous research from the ESNL has reported no significant changes in bone mineral -content, -area, or -density, or in blood markers such as calcium, alkaline phosphatase, uric acid, total protein, or creatinine when consuming a higher protein diet for 10-weeks (accompanied with resistance training), specifically in women [121].

The aforementioned literature search returned 51 studies on increased protein consumption that met the criteria for this review. Sixteen of these studies incorporated a prescribed exercise program, and will be discussed separately. Of the 35 studies not including physical activity guidelines, each study included a “higher protein consumption” group that assigned a protein intake ranging from 25-35% of daily caloric intake. Collectively, these studies demonstrate that replacing some CHO with additional PRO, can significantly decrease TG [122-139], BP [133, 138, 140-143], fasting glucose [127, 128, 134], weight [131, 134, 135, 139, 142, 144, 145] and body fat [122, 125, 131, 142, 146, 147] and increase HDL [123, 126, 131, 132, 134-136, 139], as well as decrease TC [123, 129, 133, 145, 146] and LDL [123, 129, 133, 145, 146]. Two studies even demonstrated a proven decrease in MetS prevalence [139, 148]. Replacing some CHO with an increase in the percentage of PRO seems to be a prevalent and effective treatment for all markers of MetS. These studies are summarized in Table 2.4.

Table 2.4: Increased Protein Consumption for Treatment of Metabolic Syndrome Risk Factors

Authors (Date)	Study Length Study Population	Groups	Dietary Protocol (%PRO:CHO:FAT)		Wt	BP	TG	HDL	Glu	Other
Layman et al (2002) [122]	10 weeks	CHO	CHO/PRO ratio: 3.5 (68 g pro/d)	isoenergetic; 1700kcal/d with ~50 g/d fat (<30%)	-8.1%	---	NS	NS	---	Body Fat -12.2%
	24 overwt fem; 50.1 y/o, BMI~30.3	Protein	CHO/PRO ratio: 1.4 (125 g pro/d)		-8.9%		-21.5%*			Body Fat -14.4%*
Parker et al (2002) [146]	12 weeks total; 8 wk restriction 4 wk balance	LP	16:55:26	8 weeks 1600 kcal; 4 weeks energy balance	-5.3%	NDE	-10.6%	NE	-5.5%	Men lost > fat TC: -0.2% LDL +2.8%
	54 obese (35 fem) with T2DM; ~61.2 y/o, BMI~34	HP	28:42:28		-5.6%		-16.8%		-8.8%	women lost > fat TC -6.8%* LDL -5.7%*
Volek et al (2003) [123]	4 week period; cross-over design 4 week wash out	low fat	20:55:25	Energy levels assigned to nearest 200 kcal increment based on REE from indirect calorimetry	NDE	---	NS	NS	---	NS
	10 healthy normolipidemic women; ~26.3 y/o, BMI~22	very low carb	30:10:60				-30.2%*			TC +15.8% LDL +14.6%
Gannon et al (2003) [124]	5 weeks; cross-over design 2-week wash out	high-protein	30:40:30	~2200 kcal/day with similar fat and fiber consumption	NS	NS	↓	NS	NS	---
	12 (2 fem) with untreated T2DM; ~61 y/o, BMI~31	control	15:55:30				---			
Johnston, Tjonn, Swan (2003) [149]	6 weeks	high carb low fat (HCLF)	15:66:19	<30% fat, low refined sugar (<10% total energy), high in fiber (>20 g/d)	-5.9%	---	NS	NS	NS	TC -12.2%
	16 healthy; bw 19-54 y/o; BMI~29	high pro low fat (HPLF)	32:41:27		-5.7%			-13.5%		TC -9.5% >satisfaction <hunger
Farnsworth et al (2003) [125]	16 weeks total 12 wk restriction 4 wk balance	high protein (HP)	27:44:29	12 week energy restriction (6-6.3 MJ/d) 4 week energy balance (~8.2 MJ/d)	men: - 9.7% women: 7.9%	NS	-23%*	+5%	NS	women maintained LBM*
	57 overwt/obese (43 fem) fasting insulin > 12 mU/L; ~50 y/o, BMI~33	standard protein (SP)	16:57:27				-10%			LBM -3.5%

Table 2.4: Continued

Authors (Date)	Study Length Study Population	Groups	Dietary Protocol (%PRO:CHO:FAT)		Wt	BP	TG	HDL	Glu	Other
Foster et al (2003) [126]	1 year	low-carb / high-pro	20g/day CHO initially, gradually increased	high-pro, high-fat Atkins program	-4.4%	NS	-17%*	+11%*	NS	TC NS LDL NS
	63 obese subjects (43 fem); ~44 y/o, BMI~34	conventional	15:60:25	1200-1500 kcal/day for women 1500-1800 kcal/day for men	-2.5%		+0.7%	+1.6%		
Gannon, Nuttall (2004) [127]	5 weeks; cross-over design 5-week wash out	control	15:55:30	2,825 kcal/day	NS	---	-14.4%	NS	NS	---
	8 men with untreated T2DM	higher protein	30:20:50		NS		-39.4%*		-29%*	
Sharman, Gomez, Kraemer, Volek (2004) [128]	6 weeks; crossover design no wash out	very low carb	30:10:60	hypoenergetic (-2.1 MJ/d)	---	---	-44%*	NS	-6%*	TC -10.8% LDL NS
	15 men; ~33.2 y/o, BMI~34.3	low fat	20:55:25 <10% sat fat and <300 mg chol				NS		NS	TC -14.7% LDL -18%*
Brinkworth et al (2004) [150]	12 weeks intervention	low protein	15:55:30	8 weeks energy restriction (~6.7 MJ/day) 4 weeks energy balance	-5.9%	SBP -4.5% DBP - 2.9%	-9.2%	NS	-8%	---
	38 obese patients (23 fem) T2DM; ~61.8 y/o, BMI~33.5	high protein	30:40:30		-5.5%	SBP -4.4% DBP - 4.6%	-20.3%		-7%	
Brinkworth et al (2004) [151]	68 weeks total; 12 wk restriction 4 wk balance 52 wk min support	standard protein (SP)	15:55:30	energy restriction ~6.5 MJ/day energy balance ~8.3 MJ/day	-2.9%	SBP NS DBP+1.3%	NS	+15.4%	NS	HOMA - 13.8%
	58 (45 fem) obese nondiabetic with hyperinsulinemia (fasting insulin ~17.8 mU/l); ~50.2 y/o, BMI 34	high protein (HP)	30:40:30		-4.1%	SBP NS DBP - 1.4%		+16.1%		HOMA - 19.3%

Table 2.4: Continued

Authors (Date)	Study Length Study Population	Groups	Dietary Protocol (%PRO:CHO:FAT)		Wt	BP	TG	HDL	Glu	Other
Due, Toubro, Skov, Astrup (2004) [144]	6 months strict intervention	high protein (HP)	25:45:30	energy intake was ad libitum	Wt: -10.8%* WC: -10.3%*	---	NS	NS	NS	---
	50 overwt/obese (38 fem); ~39.6 y/o, BMI~30.4	medium protein (MP)	12:58:30		Wt: -6.7% WC: -4.2%					
Appel et al (2005) [129]	6 weeks; crossover design 2 week wash	carbohydrate	15:58:27	Caloric intakes were assigned based on expenditure in order to keep body weight stable.	---	SBP -6.7% DBP -5.3%	NS	-2.3	---	TC -6.1% LDL -9%
	164 adults (73 fem) pre- or HT; ~53.6 y/o, BMI~30.2	protein	25:48:27			SBP -7.2% ^a DBP -6.8% ^a	-16.2% ^{a,b}	-5.2 ^a		TC -9.8% ^{a,b} LDL -11% ^a
		MUFA	15:48:37			SBP -7.1% ^a DBP -6.2% ^a	-9.2% ^a	NS		TC -7.6% ^a LDL -10.1%
Sargrad, Homko, Mozzoli, Boden (2005) [140]	8 weeks	high-pro	30:40:30	~1300 kcal/day	-2.6%	SBP -8.1% DBP -23.4%	NS	NS	NS	LDL -14.3%
	12 (9 fem) T2DM; ~47.5 y/o, BMI~34.5	high-carb	15:55:30		-2.3%	NS		-5.1%	-18.2%	LDL NS
Luscombe-Marsh et al (2005) [152]	16 weeks total; 12 wk restriction 4 wk balance	low-fat, high pro (LF-HP)	34:37:29	energy restriction ~6000 kJ/day (30% restriction of total energy) energy balance maintained same macro comp.	-9.5%	SBP -5.4% DBP: NS	-15.9%	+10.1%	NS	HOMA -34% TC -2.9% LDL -0.7%
	57 overwt/obese (32 fem) insulin >12mU/L; ~50.2 y/o, BMI~33.8	high-fat, standard pro (HF-SP)	18:37:45							
Hodgson, Burke, Beilin, Puddey (2006) [141]	8 weeks	control	maintain usual diet		NS	SBP +1.2% DBP NS	NS	NS	-4.1%*	---
	60 HT (22 fem); ~58.6 y/o, BMI~27.7	protein	partially replace CHO-rich foods with lean red meat	qty of lean red meat substituted: if <8500 kJ/day, given 36 g/day if >8500 kJ/day, given 50 g/day		SBP -1.4%* DBP NS			+2.0%	
McAuley et al (2006) [130]	12 months total; 4 mo supervised 8 mo follow-up	high-fat (HF)	no more than 20 g/day CHO for 2 wk, increasing to 50 g/day by 8 wk		-5.6%	NS	-25.1%	+10.5%*	NS	---
	93 overwt insulin-resistant fem	high-pro (HP)	30:40:30 consuming low-glycemic carbs		-8.5%		-36.3%*	+4.1%		
		high-carb (HC)	15:55:30 aim for 25-30 g/d fiber		-4.5%		-16.5%	-1.7%		

Table 2.4: Continued

Authors (Date)	Study Length Study Population	Groups	Dietary Protocol (%PRO:CHO:FAT)		Wt	BP	TG	HDL	Glu	Other
Leidy, Carnell, Mattes, Campbell (2007) [153]	12 weeks	normal protein (NP)	.8 g/kg/d PRO 18:57:25	-750 kcal/day	-10.7%	SBP -3.5% DBP -11.1%	-9.8%	-9.5%	-7.3%*	TC -14.6% LDL -18.6% LBM -5.8%*
	46 women; ~50 y/o, BMI~30.6	high protein (HP)	1.4 g/kg/d PRO 30:45:25		-21.3%	SBP -4.6% DBP -4.4%	-21.3%	-13.8%	+1.2%	TC -16.8% LDL -17.5% LBM -3.5%
Clifton, Keogh, Noakes (2007) [154]	64 weeks total; 12 wk weight loss 52 wk follow up	high pro (HP)	34:46:20	~5600 kJ/day with <10% sat fat	-5.2%	---	-24.3%	-26.6%	-11.5%	---
	79 healthy fem; ~49 y/o, BMI~32.8	high carb (HC)	17:64:20				-9.3%	-25.6%		
Keogh et al (2008) [131]	8 weeks	very low-carb high-sat fat (LC)	35:4:61 20% as sat fat	~30% energy restriction ~6000 kJ/day for women ~7000 kJ/day for men	-7.4%*	SBP -8.3% DBP +9.5%	-31.3%*	+7.1%*	-3.5%	abd fat -19%* TC -5.6% LDL -3.1%
	99 overwt/obese + one add'l MetS risk factor (IDF), ~50 y/o, BMI~33.7	high-carb low-sat fat (HC)	24:46:30 <8% as sat fat		-6.5%	SBP -9.6% DBP -9.1%	-16.7%	NS	-3.6%	abd fat -13% TC -9.4%* LDL -9.4%*
Tay et al (2008) [132]	24 weeks	very low carb, high fat (VLCHF)	35:4:61 20% as sat fat	energy restricted ~6-7 MJ/day 30% deficit	-12.3%	SBP -9.2% DBP -6.2%	-40%*	+17.6%*	-3.2%	TC NS LDL NS
	88 (57 fem) obese +1 add'l MetS risk factor; ~50.5 y/o, BMI~33.7	high carb, low fat (HCLF)	24:46:30 <8% as sat fat		-10.5%	SBP -7.9% DBP -7.1%	-19.7%	+6%	-3.8%	TC -10%* LDL -14.1%*
Jenkins et al (2009) [133]	4 weeks	low-carb, high pro	CHO=130g/day 31:26:43	high vegetable protein and vegetable oil	-4.7%	SBP -1.9%* DBP -2.4%*	-40.2%*	-4.2%	---	TC -20.2%* LDL -20.9%*
	44 (26 fem postmen) LDL>131 mg/dL, TG <442 mg/dL, and BMI>27	high-carb, control	16:58:25	lacto-ovo vegetarian diet	-5%	---	-21.4%	-6%		TC -12.6%, LDL -13.1%
Volek et al (2009) [134]	12 weeks	carb restrict (CRD)	28:12:59	~1,500 kcal/day	-10.4%*	---	-50.7%*	+11%*	-12%*	HOMA -55.2%*
	40 atherogenic dyslipidemia; ~32.6-37 y/o, BMI ~32-33.5	low-fat (LFD)	20:56:24		-3.3%		-19.3%	-2.6%	-2.1%	HOMA -17.6%

Table 2.4: Continued

Authors (Date)	Study Length Study Population	Groups	Dietary Protocol (%PRO:CHO:FAT)		Wt	BP	TG	HDL	Glu	Other
Lee et al (2009) [147]	12 week	high protein (HP)	30:50:20	1500 kcal/day men 1200 kcal/day women 25g/day fiber meal replacement 2x/day	Wt: -6.6% WC: -6.6%	---	-29.4%	+11.2%	NS	>70% compliance: -15.9% FM*
	75 subjects; ~48 y/o, BMI~28.5	conventional (C)	15:65:20		Wt: -6.4% WC: -7.5%		-24.9%	+17.2%	-4.3%	>70% compliance: -8.7% FM
Claessens, van Baak, Monsheimer, Saris (2009) [135]	18 weeks total; 5-6 wk restriction 12 wk maintain	high-carb (HC)	weight maintenance >55% CHO	5-6 wk restriction, - 500 kcal/day liquid VLCD + unrestricted low- carb vegetables ~30% fat on wt maintenance	Wt: -8.2% WC: -9.2%	SBP -6.0% DBP -4.2%	+23.8	NS	-2.6%	---
	48 (31 fem); ~45.5 y/o, BMI~33	high-pro casein (HPC)	weight maintenance >25% PRO		Wt: -11.0%* WC: -10.6%*	SBP -6.5% DBP -8.1%	-22.9*	+10.9%	+2.9%*	no diff bw protein groups
		high-pro whey (HPW)			Wt: -11.2%* WC: -11.5%*	SBP -9.6% DBP -9.7%	-28.2%*	+9.1%	-1.8%*	
Brinkworth et al (2009) [136]	12 months	very low carb, high sat fat (LC)	35:4:61	energy restricted ~6-7 MJ/day	-15.4%	SBP -10.4% DBP -8.7%	-34.7%*	+20.7%*	-5.3%	FFM -6% TC +13.0% LDL +19%*
	69 obesity + 1 MetS risk factor; ~51.4 y/o, BMI~33.4	high carb, low fat (LF)	24:46:30	isocaloric	-12.2%	SBP -10.8% DBP -10.2%	-12.2%	+5.1%	-5.4%	FFM -4.1% TC +1.8% LDL +2.9%
Lim, Noakes, Keogh, Clifton (2009) [137]	3 months intensive support	very low carb (VLC)	35:4:61 20% as sat fat	energy content = 6500 kJ	-9.1%	SBP -8.1% DBP -4.9%	-38.9% ^a	0% ^a	-1.9%	---
	113; ~47 y/o, BMI 32, + one add'l CVD risk	very low fat (VLF)	20:70:10 3% as sat fat		-7.5%	SBP -5.5% DBP -2.7%	-6.3% ^b	-7.1% ^b	+1.9%	
		high unsat fat (HUF)	20:50:30		-6.8%	SBP -2.4% DBP -2.5%	-12.5% ^b	-7.7% ^b	-3.7%	
Papakonstantinou et al (2010) [138]	4 weeks crossover design 3 week washout	high pro, low fat (HPLF)	30:50:20	crossover between groups	Wt: -3.3% WC: -31.9%	SBP -9.0%* DBP -9.3%*	-29.4%*	-8.3%	-11.3%	TC -12.5% LDL -13.5%
	17 obese (12 fem) T2DM; ~46 y/o, BMI 31-45	low pro, high fat (LPHF)	15:50:35		Wt: -3.2% WC: -3.7%	SBP -3% DBP NS	-21.1%	NS	-13.3%	TC -10.9% LDL NS
Morenga, Williams, Brown, Mann (2010) [145]	10 weeks	standard low- fat, high-carb	20:50:30	advice regarding strict adherence to energy intake goals was not given.	Wt: NS WC: NS	NS	NS	NS	NS	TC: -3.3% LDL: +2%
	89 overwt/obese fem; ~42 y/o, BMI~32.7	high pro, high fiber (HPHF)	30:50:20 >35 g/d fiber		Wt: -1.8%* WC: -2.3%				-2.1%	TC: -4.6%* LDL: -5%*
Lopez-Jimenez, Xu, Edens (2010) [148]	6 months	experimental	lower CHO, relatively high protein MUFA and PUFA enriched		-6.4%	---	-24.4%	+3.8	-1.4%	-32%* MetS Prevalence
	55 MetS patients (ATP III) BMI ~34-35	control	<30% fat	standard rec for diabetics	-4.7%		-21.1%	-0.4%	-1.5%	-15% MetS Prevalence

Table 2.4: Continued

Authors (Date)	Study Length Study Population	Groups	Dietary Protocol (%PRO:CHO:FAT)		Wt	BP	TG	HDL	Glu	Other
Flechtner-Mors et al (2010) [139]	12 months	conventional protein (C)	15:55:30 .8g/kg body wt	500 kcal/d less than estimated RMR	Wt: -6.62% WC: -7.2	---	-5.4%	0%	-10.2%	34.8% no longer met MetS criteria
	110 (88 fem) with MetS; ~49.75 y/o, BMI~36.25	high pro (P)	30:40:30 1.34 g/kg		Wt: -9.12%* WC: -10.9*		-34.7%*	+4.6%*	-9.1%	64.5% no longer met MetS criteria
Toscani et al (2011) [155]	8 weeks	high protein (HP)	30:40:30	Energy content estimated to 20-25 kcal/kg current weight per day	P: Wt: -4.3% WC: -2.0% C: Wt: -1.8% WC: -2.5%	NS	NS	NS	NS	---
	30 (18 with PCOS [P]) ~22.7 y/o, 22 controls [C] ~29.3 y/o) BMI >25	normal protein (NP)	15:55:30		P: Wt: -3.7% WC: -3.5% C: Wt: -4.1% WC: -2.8%					
Morenga et al (2011) [142]	8 weeks	mod-high pro (HP)	30:40:30	-2000-4000 kJ/day for a 0.5-1 kg/wk weight loss	Wt: -4.9%* WC: -5.8	SBP -4.3% DBP -5.9%*	-20.7%	NS	-38%	body fat - 6.1%*
	83 overwt/obese fem; ~41.9 y/o, BMI~33.9	high-fiber high- carb (Hfib)	20:50:30 >35 g/d fiber		Wt: -3.6% WC: -4.8%	SBP -1.4% DBP -1.1%	-12.9%	NS	-53.1%	body fat - 3.3%
Pearce, Clifton, Noakes (2011) [156]	12 weeks	high-pro low-chol (HPLchol)	213 mg cholesterol	hypoenergetic (6-7 mJ) 30:40:30	-6%	SBP -3.5% DBP -4%	-23%	-5.6%	-9.3%	TC -5.8%
	65 T2DM or IGT; ~54.4 y/o, BMI 34.1, LDL 2.67 mmol/l	high-pro high- chol (HPHchol)	590 mg cholesterol			SBP -7.7% DBP -8.5%	-25%	+1.5%*	-4.4%	TC -1.5%
Larsen, Mann, Maclean, Shaw (2011) [143]	3 month energy restriction	high protein	30:40:30	3 month energy restriction ~6400 kJ/day or 30% energy reduction	Wt: -2.9% WC: -2.8%	SBP -2.3%* DBP NS	-20.9%	12 mo: +6.7%	---	TC -4.9%
	99 overwt/obese (51 fem) T2DM; ~59.3 y/o, BMI 27-40	high carb	15:55:30		Wt: -3.2% WC: -2.3%	SBP 0% DBP NS	-19%	12 mo: +6.7%		TC -6.6%
BMI (body mass index) values are listed in kg/m ² . Abd, abdominal; ATP III, National Cholesterol Education Program - Adult Treatment Panel III; BP, blood pressure; CHO, carbohydrate; DBP, diastolic blood pressure; Fem, female; FFM, fat free mass; FM, fat mass; Glu, glucose; HDL, high density lipoprotein cholesterol; HOMA, homeostatic model assessment; HT, hypertension; IDF, International Diabetes Foundation; LBM, lean body mass; LDL, low density lipoprotein cholesterol; MetS, metabolic syndrome; MUFA, monounsaturated fatty acids; PCOS, polycystic ovarian syndrome; PRO, protein; PUFA, polyunsaturated fatty acids; RMR, resting metabolic rate; SBP, systolic blood pressure; T2DM, type 2 diabetes mellitus; TC, total cholesterol; TG, triglycerides; VLCD, very low calorie diet; WC, waist circumference; Wt, weight; y/o, years old. Values are calculated percent change from baseline. Listed values are significantly different from baseline, representing a time effect. Bold and * indicate p<0.05 for group/diet effect. Within a study, values that do not share a common superscript are significantly different. ---, value not reported or measured; NS, no significant change from baseline; NDE, no diet effect; ⬆️⬆️ significant change, yet specific value not reported.										

While there is agreement that total energy intake is the primary focus of weight loss and management, the dispute regarding the proper macronutrient balance is still a concern. Research by Campbell and Meckling [67] has shown that while the amount of weight loss may be similar regardless of the macronutrient composition, a higher protein diet may result in more fat loss and lower fasting insulin concentrations. Additionally, a review on the differential effects of dietary treatments on blood lipids has suggested that there may be benefit in tailoring treatments to specific individuals in such that a CHO-based, low-fat diet may work better for persons needing to lower total cholesterol (TC) and LDL, while a PRO-based diet could positively benefit individuals who have elevated TG and low HDL [43]. Perhaps the same dietary prescription will not work for all health profiles.

Physical Activity

Physical activity may be a key player in the cause and treatment of MetS. Mokdad et al [157] suggest that the combination of poor diet and inactivity could become the leading cause of preventable death in the US, overtaking tobacco use. Bassuk et al [46] have estimated that only 11-46% of the US adult population engages in the recommended amount of physical activity regularly. It is projected that as much as 70% of the US population is classified as sedentary (defined as less than three 20-minute sessions of demanding physical activity a week) [33, 60, 71]. Additionally, women may not be as likely to reach these recommendations as men (47.9% and 50.7% respectively) [47].

Low activity levels are related to the individual components of MetS [5], so much so that a dose-response relationship is identified between low physical activity levels and increased CVD risk [44, 45]. Physical inactivity can lead to CVD risk in a similar manner as obesity, by increasing inflammatory markers and fasting plasma glucose levels, as well as elevating blood pressure [158]. Additionally, a sedentary state can lead to obesity, modified insulin sensitivity in the muscle, and an increased risk of diabetes [77, 159]. Conversely, being physically active could possibly reduce the risk of developing diabetes (by as much as 34% for every one hour of brisk walking per day) [77, 159].

General Physical Activity

Moderate physical activity can improve the risk factors and/or reduce the likelihood of developing MetS [8, 49]. Over time, increased activity could lead to lower body weight and higher cardiorespiratory fitness levels, both of which would modulate insulin action and other components of MetS [17, 45, 51, 60]. Specifically, physical activity is associated with reductions in resting BP, abdominal fat, TG, and fasting glucose, as well as an increase in HDL cholesterol [24]. While the current guidelines recommend participating in moderate-intensity exercise for ≥ 30 minutes on most days of the week [1, 47], specific studies have found higher fitness levels and more vigorous activity (5-9 metabolic equivalents) to be the most strongly correlated with MetS risk reduction [13, 45, 160, 161]. Others have found both the amount of physical activity (expenditure) and fitness level (measured by peak oxygen consumption, $\text{VO}_2 \text{ max}$) to have independent affects on MetS components [45, 162, 163], while some question the

significance of the correlation with VO_2 max [163]. Additional findings have proven that even in the absence of enhanced fitness, physical activity is still beneficial in creating a more desirable metabolic profile [52, 164].

Aerobic Activity

Extensive research has proven cardiorespiratory fitness to be inversely correlated with the metabolic abnormalities of MetS, CVD, and T2DM regardless of age, gender, ethnicity, or body weight [3, 6, 14, 24, 29, 50-53]. Specifically, the Cross-Cultural Activity Participation Study by Irwin et al [56] found the odds of having MetS to be 82% lower among the most active women, 86% lower among women reporting any amount of vigorous activity, and 93% lower among women in the highest quartile of maximum treadmill duration compared to their lowest level counterparts. Similarly, individuals in the lowest fitness category may be ten times as likely as those in the highest fitness category to develop MetS [24]. In regards to specific cardiovascular measurements, Okura et al [52] found each one-ml/kg/min increase in VO_2 max to translate to ~7% reduced risk of MetS. LaMonte et al [165] computed a one-metabolic equivalent increase in maximal treadmill testing to equate to a ~17% reduced risk level, specifically in women. More precisely, it has been calculated that a woman in her 40's would need to achieve a VO_2 of ~28 ml/kg/min in order to reduce her MetS risk by moving out of the lowest fitness category [24].

The ability to drastically increase cardiorespiratory fitness is questionable based on genetic and environmental limitations [24]. The appropriate exercise prescription and intensity is also heavily debated [6]. Some researchers believe that lower intensity (40-

60% VO₂ max) exercise that can be maintained for a longer duration (≥ 60 minutes), more frequently (up to five days a week) will lead to the greatest improvement in cardiorespiratory fitness [51]. Moderate and moderate-vigorous ($>60\%$ VO₂max) intensities have also been purported to be most effective [166]. Previous reviews [51, 53, 54] have found that while both moderate and vigorous activity may equally affect body fat, systolic blood pressure (SBP), and lipid profile (lowering TG and raising HDL), vigorous intensity may be superior for further affecting diastolic blood pressure (DBP) and glucose metabolism. Although benefits may be achieved with either intensity, shorter time periods with more vigorous activity could be utilized [166]. A review by Laaksonen et al [167] stated that 180 minutes/week of moderate intensity aerobic training, but only 60 minutes of moderate-vigorous activity, is necessary to decrease the risk of MetS by 50% [167]. It is promising to note that even a single bout of activity, either moderate or vigorous intensity, can promote short-term benefits to include lower TG, SBP and DBP, and greater insulin sensitivity [168].

Resistance Training

Resistance exercise appears to produce risk-reducing benefits as well. It is postulated that resistance training may provide protective effects through the achieved strength gains, reduction in body fat, and improvement of insulin sensitivity [21, 42, 47]. Muscular strength has been suggested to be inversely associated with all-cause mortality [47] and reduced risk of developing MetS (by 34%) [169, 170]. Previous reviews have found routine resistance training (2-3 times/week) to lead to decreased resting BP,

abdominal fat, and improved insulin resistance [53, 170], but limited to no benefit has been reported on fasting lipid levels [170].

When combined with a hypocaloric diet, resistance training can aid in the maintenance and/or increase of both lean body mass and resting metabolic rate, which may otherwise be at risk of decrement with diet alone [62, 170]. There is a progressive reduction in strength and muscle mass associated with aging, which may explain the increasing risk of conditions such as MetS over the life span. Since RT has proven to be more effective in the prevention rather than the restoration of muscle mass [118], performing this type of activity at an early age, and throughout life, may offset the deleterious effects. Finally, further benefit may be achieved by the combination of aerobic and resistance training exercises, with aerobic exercise incorporating the improvements to the lipid profile as well [47, 171].

Protein Modification with Exercise

As the independent benefits of both higher protein consumption and exercise have each been previously discussed, the combination of the two may lead to even greater prevention and/or reversal of MetS. It is known that the combination of exercise with a higher protein diet can enhance weight loss, fat loss, and aid in fat free mass retention [43], which is certainly beneficial for an individual with MetS.

Sixteen studies that incorporate higher PRO consumption with physical activity met the criteria for this literature review, and are summarized in Table 2.5. These studies utilize PRO percentages ranging from 22-34% of daily caloric intake.

Table 2.5: Increased Protein Consumption with Exercise for Treatment of Metabolic Syndrome Risk Factors

Authors (Date)	Study Length Study Population	Groups	Dietary Protocol (%PRO:CHO:FAT)		Exercise	Wt	BP	TG	HDL	Glu	Other
Suh and Lee (2005) [172]	8 weeks	high-pro + exercise (HPE)	25% caloric intake was soy protein		aerobic activity 3x/week for >30 min @ 50-65 max capacity	-4.1%*	---	NS	+5.8%*	-9.8%*	TC: -7.8%*
	30 fem; ~24.5 y/o, BMI~21.9	exercise only (EXO)	maintain usual diet			-3.3%		NS	NS	NS	TC: NS
		control (CON)				---		NS	NS	NS	TC: NS
Noakes, Keogh, Foster, Clifton (2005) [173]	12 weeks	high-pro (HP)	34:46:20	Isocaloric 5600 kJ	increase activity to ≥30 min 3x/week	-8.75%	---	-21.9%*	-6.8%	-3.4%	---
	100 fem; ~49 y/o, BMI~32	high-carb (HC)	17:64:20		-8.05%	-8.05%	-7.5%	-6.8%	-4.1%	-4.1%	
Layman et al (2005) [43]	4 month	CHO	.8 PRO: >3.5 CHO g/kg/day	both diets 7.1 MJ/d and 30% fat	voluntary, walking 30 min 5d/wk	-8.3%	---	NS	-7.7%	---	TC: -10.1% LDL: -13%* > FFM loss
	48 fem, ~46 y/o, BMI~33	CHO + EX	1.6 PRO: <1.5 CHO g/kg/day		mandatory 5 d walk 2 d RT/wk	-8.4%		NS	NS		TC: -9.0% LDL: -10%*
		HP + Ex			voluntary, walking 30 min 5d/wk	-11.4%*		-25.2%*	NS		TC: NS LDL: NS
		HP				-9.5%*		-21.1%*	NS		TC: NS LDL: NS
McAuley et al (2005) [174]	24 weeks total; 8 wk weight loss 8 wk maintenance 8 wk no supervision	high-carb, high-fiber (HC)	focused on number of servings of each food group	recommendations involved food choices, did not prescribe a total energy amount for consumption	encouraged to exercise 30 min 5x/wk	Wt: -4.8% ^a WC: -6.3% ^a	SBP -1.6% DBP -1.3%	-18.1% ^a	-3.4% ^a	-6%	---
	96 normoglycemic, insulin resistant fem, 30-70 y/o, BMI >27	(HF) high-fat / Atkins Diet	20g/day CHO gradually increased			Wt: -7.4% ^b WC: -9.0% ^b	SBP -3.1% DBP -2.4%*	-39.9% ^b	+7.7% ^b	-5.9%	
		high-pro / Zone Diet (HP)	30:40:30			Wt: -7.4% ^b WC: -8.1% ^b	SBP -2.4% DBP -1.3%	-31.2% ^b	+0.8% ^{a,b}	-3.9%	
Dansinger et al (2005) [175]	2 months strict intervention	Atkins	<20g/d CHO with a gradual increase to 50 g/d CHO		encouraged to perform 60 min weekly	Wt: -3.6% WC: -3%	SBP: -3.3% DBP: -5.5%	-15.1%*	+6.7%*	-7.7%	TC: NS LDL: NS
	160 overweight/obese known HT, dyslipidemia, or hyperglycemia; BMI~35	Zone	30:40:30			Wt: -3.8% WC: -2.8%	SBP: NS DBP: -6.2%	-24.4%*	NS	NS	TC: -8.3%* LDL: -7%*
		Weight Watchers	24-32 points daily; ~1,200-1,600 kcal/day			Wt: -3.6% WC: -3.2%	SBP: -3.6% DBP: -4.2%	NS	NS	TC: - 7%* LDL: - 9%*	TC: -6.7%* LDL: -9%*
		Ornish	vegetarian diet with <10% energy from fat			Wt: -3.5% WC: -2.4%	SBP: NS DBP: -3.3%	NS	-8%	TC: - 9%* LDL- 12%*	TC: -8.9%* LDL: -12%*

Table 2.5: Continued

Authors (Date)	Study Length Study Population	Groups	Dietary Protocol (%PRO:CHO:FAT)		Exercise	Wt	BP	TG	HDL	Glu	Other
Ferrara et al (2006) [176]	6 months	high pro (HP)	22:50:28	1.9 g/kg PRO	performing 90+ min an-/aerobic training 3x/wk	-2.8%	NS	NS	NS	NS	TC -25%
	15 healthy men; ~26.4 y/o, BMI~23.5	normal protein (NP)	15:60:25	1.3 g/kg PRO		NS					TC -15.3%
Meckling and Sherfey (2007) [120]	12 Weeks	CON	1:1 PRO:CHO ≤30% fat	500 kcal/d less than analyzed intake	---	-2.7%	SBP -7.1% DBP -7.4%	NS	NS	NS	TC NS LDL NS
	44 overwt/obese fem; ~42.5 y/o, BMI~30	CONEx			3x/week, 36 min circuit, 65-80% MHR	-5%	SBP -5.4% DBP NE				TC -17.5% LDL NS
		HPEX	3:1 PRO:CHO (~.75g/kg PRO; ~1371 kcal/d)		---	-8.1%	SBP -5.2% DBP -4.9%	-29.9%*			TC NS LDL NS
		HP				-5.5%	SBP -7% DBP -8.9%	NS			TC -32% LDL -41.5%
Bowden et al (2007) [177]	12 weeks	same kcal (Diet 1)	same caloric consumption as baseline	High-pro / low-carb, 25:45:30	recommended 30 min/day 4-6 x/wk @ 60-85% MHR	NS	---	NS	NS	---	DEXA BF -6.86%*
	48 young, normolipidemic, normoglycemic, sedentary	-500 kcal (Diet 2)	-500 kcal/day from baseline						↑		NS
Gardner et al (2007) [178]	12 months total: 2 mo weekly instruction 10 mo follow up	Atkins	20 g/day CHO gradual increase to 50 g/day	no focus on energy restriction	some encouragement to increase activity	-5.2%^a	SBP -6.4%^a DBP -6%^a	-23.4%^a	+9.2%^a	NS	---
		Zone	30:40:30	incorporated specific goals for energy restriction		-1.7% ^b	SBP -2.9% ^b DBP -3% ^{a,b}	-3.4% ^b	+4.2% ^{a,b}	NS	
	311 overwt/obese, nondiabetic, premenopausal; ~41 y/o, BMI~32	LEARN	55-60% CHO, <10% sat fat			-3.0% ^{a,b}	SBP -2.7% ^b DBP -3% ^{a,b}	-12.3% ^{a,b}	+5.5% ^{a,b}	NS	
		Ornish	10% fat	no focus on energy restriction		-2.4% ^{a,b}	SBP -1.6% ^b DBP -1% ^b	-12.6% ^{a,b}	0% ^b	NS	

Table 2.5: Continued

Authors (Date)	Study Length Study Population	Groups	Dietary Protocol (%PRO:CHO:FAT)		Exercise	Wt	BP	TG	HDL	Glu	Other
Lasker, Evans, Layman (2008) [179]	4 month	PRO	~1700 kcal/day 30:40:30	1.6 g/kg/d Pro <170 g/d CHO	~90 min/week	-9.1% (p=0.07)	---	-34%*	+5%*	NS	FM: -8.7%* LDL: +2.5%
	50 subjects; ~47 y/o, BMI ~33.6	CHO	~1700 kcal/day 15:55:30	0.8 g/kg/d PRO >220 g/d CHO		-7.3%		-14%	-3%		FM: -5.7% LDL: -7%*
Layman et al (2009) [180]	4 month wt loss	moderate pro (PRO)	1.6g/kg/d PRO <170 g/d CHO (30:40:30)	1700 kcal/day females; 1900kcal/d males; 30% fat; 17 g fiber	voluntary, walking 30 min 5d/wk	-8.7%,	---	↓	↑	---	FM -22%
	130 subjects; ~45.4 y/o, BMI ~32.6	conventional high-carb (CHO)	0.8 g/kg/d PRO >220 g/d CHO (15:55:30)	-7.6%,		---		---	TC ↓ LDL ↓		
Sacks et al (2009) [181]	2 years	low-fat, ave-pro	15:65:20	-750 kcal/day deficit from baseline intake with 20 g/day dietary fiber, <150 mg/cholesterol per 1000 kcal, <8% saturated fat	90 min/wk of moderate activity	NS	SBP -0.8% DBP -0.8%	-11.5%	+5.6%	+1.1	LDL: -5.9%*
	811 overwt; ~51 y/o, BMI ~33	low-fat, high-pro	25:55:20				SBP -1.7% DBP -1.3%	-16.6%	+6.5%	+1.0	LDL: -3.9%*
		high-fat, ave-pro	15:45:40				SBP -1.3% DBP -1.5%	-12.4%	+6.3%	+1.6	LDL: -0.2%
		high-fat, high pro	25:35:40				SBP -0.7% DBP -0.3%	-16.7%	+8.8%*	+2.8	LDL: -1.3%*
Wycherley et al (2010) [42]	16 weeks	Con	19:53:26	Females 6 MJ/day Males 7 MJ/day	---	Wt: -8.9% WC: -7.4%	SBP -9.5% DBP -8.9%	-26.1%	0	-23.9%	---
	83 with T2DM, ~56.1 y/o, BMI~35.4	Con + RT			RT 3x/week, 2 sets, 8-12 reps @ 70-85% 1RM	Wt: -10% WC: -9.9%	SBP -12% DBP -9.9%	-18.8%	-9.1%	-21.8%	
		HP + RT	33:43:22		---	Wt: -13%* WC: -12%*	SBP -10% DBP -8.9%	-27.8%	-9.1%	-23.2%	
		HP				Wt: -8.8% WC: -7.8%	SBP -11% DBP -12%	-20%	-8.3%	-26.3%	
Josse et al (2011) [182]	16 weeks	high pro, high dairy (HPHD)	30:40:30	30% total pro, 15% from dairy	7 d/wk aerobic exercise for 250 cal. expenditure; 2 d/wk RT	Wt -5% WC -4.9%	---	↓	---	---	FFM +1.4%* TC: ↓ LDL: ↓
	90 overwt/obese premeno fem; ~28 y/o, BMI ~31	adeq pro, med dairy (APMD)	15:55:30	15% total pro, 7.5% from dairy				---			FFM -0.4%
		adeq pro, low dairy (APLD)	15:55:30	15% total pro, <2% from dairy				↓			FFM -1.4% TC: ↓ LDL: ↓

Table 2.5: Continued

Authors (Date)	Study Length Study Population	Groups	Dietary Protocol (%PRO:CHO:FAT)		Exercise	Wt	BP	TG	HDL	Glu	Other
Campbell and Meckling (2012) [67]	12 weeks	LP	1g PRO: 4 g CHO	low fat, hypo- and iso-energetic	60 minutes, circuit, 3x/week @ 65-80% MHR	Wt: -6.3% WC: -7.6% ^{aa}	SBP -6.7% DBP -6.7%	-15.1%	-13.7%	NS	lean mass +4.2%
	54 overweight/obese women with MetS risk factors; (~40.2 y/o, BMI~35.5 kg/m2	NP	1g PRO: 2g CHO			Wt: -9.1% WC: -11%^{ab*}	SBP -6.3% DBP -7.2%	-26.6%	-16.3%		lean mass +10.0%*
		HP	1g PRO: 1g CHO			Wt: -7.2% WC: -8.1% ^b	SBP -7.9% DBP -6.3%	-18.0%	-12.6%		lean mass +7.3%
Dutheil et al (2012) [183]	26 weeks total; 3 weeks residential 6 months follow up	Normal protein intake (NPI)	1.0 g/kg/day	-500 kcal/day	2-3 hrs/day (2 hr walking, 1 hr lt aerobics) 40-60% HRR	Wt: -5.4% WC: -5.9%	NS	NS	NS	NS	TC NS LDL NS
	28 (9 fem) with MetS (IDF); 61.8 y/o, BMI~33.4	high protein intake (HPI)	1.2 g/kg/day			Wt: -8.2% WC: -8.2%					
BMI (body mass index) values are listed in kg/m ² . BF, body fat; BP, blood pressure; CHO, carbohydrate; DBP, diastolic blood pressure; FFM, fat free mass; FM, fat mass; Glu, glucose; HDL, high density lipoprotein cholesterol; HRR, heart rate reserve; HT, hypertension; LDL, low density lipoprotein cholesterol; MetS, metabolic syndrome; MHR, maximum heart rate; PRO, protein; RT, resistance training; SBP, systolic blood pressure; T2DM, type 2 diabetes mellitus; TC, total cholesterol; TG, triglycerides; WC, waist circumference; Wt, weight; y/o, years old. Values are calculated percent change from baseline. Listed values are significantly different from baseline, representing a time effect. Bold and * indicate p<0.05 for group/diet effect. Within a study, values that do not share a common superscript are significantly different. ---, value not reported or measured; NS no significant change from baseline; NDE, no diet effect; éé significant change, yet specific value not reported.											

While most studies are comparisons with a higher carbohydrate consumption group, some studies compared popular diet programs (such as Atkins, Zone, and Weight Watchers) and others compared different protein allotments. Most of the studies focused on increasing aerobic exercise, while a few utilized resistance exercises as well. While the studies that integrated resistance training may have further aided in weight loss [42, 43] and lean mass gain [182], both aerobic and resistance training (combined with a higher protein diet) lead to similar benefits regarding MetS: decreased TG [43, 120, 173, 174, 178-180, 182] and weight [42, 43, 172, 174, 178], increased HDL [172, 175, 178-181], and a few studies also found decreases in glucose [172], and blood pressure [178], as well as fat mass [179, 180] and TC [172]. Perhaps more differences between the types of exercise would have been noted if looking at fitness variables (such as strength, muscular endurance, and cardiorespiratory fitness) however this review focused specifically on the markers of MetS.

Weight Loss

Based on the research reviewed above, weight loss appears to be an integral component in the treatment of MetS [33, 55, 56]. More drastically stated, a linear relationship has been found to exist between weight and WC, SBP, DBP, TG, HDL, and fasting glucose [50, 57]. In a study by Hillier et al [57], each kilogram gained resulted in a 22% increase in risk of developing MetS. A decrease in body weight has been found to lower TC, TG, and glucose levels, decrease BP, and reduce insulin resistance [58]. Weight reduction will not only improve each of the MetS risk factors, it will also specifically reduce the risk for T2DM [8]. Surprisingly, it only takes a small reduction

to achieve health benefits. The National Institutes of Health has shown that losing as little as five-to-ten pounds can decrease elevated glucose levels [21, 184]. A decrease of seven-to-ten percent body weight over 6-12 months is advised for the treatment of MetS [27, 58-60]. It is promising that ideal body weight need not be achieved in order to begin the reversal of the diseased state.

The benefits of a TLC program with diet and exercise collectively, versus either component individually, have been repeatedly proven. Ross et al [33, 185] have shown that while both diet-induced and exercise-induced weight loss may lead to similar decrements in abdominal obesity and insulin resistance, cardiovascular exercise is beneficial in improving fitness and reducing abdominal fat specifically. Layman et al [43] have found the effects of diet and exercise combined to be independent and additive in their improvement of both absolute and percent change in body fat. Cardiovascular exercise is beneficial in maximizing caloric expenditure, while resistance training is key to maintaining or increasing lean body mass particularly in conjunction with a deficit in caloric consumption [67].

With various compositions of the MetS profile, perhaps the same protocol for weight loss will not be advantageous for each patient [26]. Depending on the exact health concerns requiring attention, general body weight loss can be beneficial for insulin sensitivity and lipid metabolism, while loss of fat mass specifically, may be necessary for blood pressure reduction [55].

INDIVIDUAL RISK FACTORS

A review of each NCEP ATP III MetS risk factor independently is beneficial toward the development of individualized TLC programs. These risk factors will be discussed in the order of their prevalence in women according to the 2003-2006 NHANES report: abdominal obesity (53%), hypertension (40%), hyperglycemia (39%), hypertriglyceridemia (31%) and low HDL cholesterol (25%) [19].

Obesity

According to the NCEP ATP III definition, a waist circumference (WC) >88 cm is a risk factor for MetS in women [8]. Central obesity is heavily interrelated with additional risk factors of MetS, CVD, and T2DM, and according to some may be the driving force behind the increased MetS prevalence [1, 5]. Past research has shown WC alone to be an independent predictor for CVD [42, 83, 186, 187]. Abdominal obesity is linked to metabolic abnormalities such as elevated fasting glucose, blood pressure, and inflammatory markers [158]. It is closely associated with insulin resistance (which is a powerful risk factor in the development of T2DM and CVD) [17], and has the potential to cause an atherogenic lipoprotein profile (to include hypertriglyceridemia and reduced HDL, among other lipoprotein abnormalities) [85]. Health conditions related to obesity are to blame for over 300,000 US deaths and \$117 billion in health care expenses each year [21, 22].

Obesity Treatment

Weight reduction is essential for reversing MetS, and should be achieved through a combination of reduced caloric intake and increased physical activity [67]. Due to the

thought that excess dietary fat may be to blame for obesity, the American Heart Association among other organizations advise a low-fat diet [67]. Interestingly, others have pointed out that the prevalence of MetS has increased even in conjunction with a decrease in dietary fat consumption in the US [67, 188]. The vague guidelines for reducing a large WC in MetS have included weight reduction, consumption of a hypocaloric diet, increased physical activity, and utilization of drugs if necessary [17]. While Buchholz and Scheoller [189] firmly believe that “a calorie is a calorie” in regards to weight loss, research from the ESNL has shown that greater PRO consumption, when in combination with a resistance-based circuit training program, may be more effective than higher CHO ingestion for WC reduction [62, 63, 83]. Additional studies have professed the benefits of utilizing both aerobic training for reducing body mass and fat mass and resistance training for increase lean mass and potentially decrease fat mass as well [171].

Blood Pressure

Blood pressure higher than 135/80 mmHg (in addition to taking blood pressure medication) is a risk factor for MetS according to the NCEP ATP III [8]. According to the Center of Disease Control [190], one-in-three US adults (68 million people) have high blood pressure. Additionally the American Heart Association stated in 2012 that 30% of Americans have prehypertension [191]. A pre-hypertensive diagnosis increases the risk of developing hypertension (HT), and hypertension increases risk for CVD [192]. The good news is that TLC works well for mild hypertension [5], and a focus on weight loss is also beneficial towards blood pressure reduction [193]. This relationship

is achieved primarily through increases in blood volume and cardiac output as well as other physiological changes in the renin-angiotensin system [58, 194, 195]. Ramsey et al [58, 194, 195] anticipate that every one-kilogram reduction in weight loss may result in a 1.5-2.5 mmHg drop in blood pressure. Additionally, a large meta-analysis has found that lowering systolic (SBP) and diastolic (DBP) blood pressure by ten and six mmHg respectively, can lead to a 40% and 30% reduction in the risk of cardiovascular-related premature death [13, 196].

Blood Pressure Treatment

In addition to the general recommendation of weight reduction [17], the following recommendations have been suggested for the treatment of elevated blood pressure with MetS: dietary salt restriction [17, 58], increased potassium intake [58], and medication as needed. The Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure states in their guidelines that drug therapy is required in patients with BP >140/90 mmHg [17, 197]. However, utilization of particular hypertensive medications (such as TZDs and beta blockers) may exacerbate other risk factors of the metabolic syndrome [17, 73, 198].

In regards to diet and exercise, there is not conclusive evidence on the efficacy of ingesting any particular macronutrient for BP reduction [115]. A review by Eriksson et al [5] reported the following regarding exercise: it can provide a moderate antihypertensive effect, it is most beneficial in cases of mild hypertension or prevention, it can prevent age-associated elevations in BP, and that these benefits of exercise may be greater in women than in men. Additionally, the antihypertensive effects of exercise may

be due to the accumulation of single exercise bouts rather than chronic changes, although long-term resistance training can favorably lower resting BP. One meta-analysis has even estimated that exercise can be utilized to decrease SBP and DBP by eleven and eight mmHg respectively [199].

Hyperglycemia

As an easy and effective tool for measuring hyperglycemia, the NCEP ATP III recommends a fasting plasma glucose test, where a value ≥ 5.6 mmol/L is an indicator for MetS [8]. The Center of Disease Control estimated that as of 2003, 14.4% of US adults (29 million people) have diabetes or impaired fasting glucose [200].

Hyperglycemia alone is not responsible for the increased CVD risk; rather it is the combination with the metabolic abnormalities that accompany it [85, 201]. Elevated glucose suggests a prediabetic state, and may be a predictor of insulin resistance. Lipid overload specifically in the liver and muscle tissues may be to blame, which could lead to a suppressed insulin regulation of hepatic glucose output and impaired insulin-mediated glucose disposal respective to each tissue [28, 51]. Grundy et al [28] define the three major causes of insulin resistance to be genetics, abdominal obesity, and lack of exercise. Additionally, obesity, hypertension, dyslipidemia, and a sedentary lifestyle are all factors related to MetS that can also predispose an individual to insulin resistance, reducing the target tissue's ability to respond to normal concentrations of insulin [21].

There are many mechanisms that could be responsible for linking insulin resistance to CVD; to include increased free fatty acid levels, increased sympathetic nervous system activity, vascular smooth muscle cell proliferation, impaired endothelial

function, and inflammation, although the specific cause(s) of insulin resistance are not well understood [71, 202, 203]. Obesity causes grave concern towards the development of insulin resistance (as well as T2DM and CVD) as the accumulation of fat mass has been found to drive insulin resistance, and insulin sensitivity decreases as body fat percentage increases [28, 42, 85]. Abdominal fat in particular leads to higher levels of free fatty acids than lower body fat accumulation, which may explain the linkage between insulin resistance and abdominal obesity [28].

Hyperglycemia Treatment

Body weight and physical fitness both have the ability to regulate insulin resistance and reverse hyperglycemia [45, 48, 204]. The National Institutes of Health has found that losing as little as five-to-ten pounds may lower elevated glucose levels into a healthy range [21, 205]. Exercising regularly will lead to an increase in insulin sensitivity in the skeletal muscles [118, 206]. Some short-term benefits of physical activity include an increase in the number of muscular glucose transporters and improved insulin-mediated glucose disposal. Recurrent exercise can lead to reduced free fatty acid levels and increased insulin sensitivity [5, 28, 56, 170, 207]. Additionally, fitness level (VO₂max) has been correlated with the prevention of T2DM, with low fitness levels leading to an increased risk [208].

Generally stated, “dietary modification and enhanced physical activity” could prevent the evolution of prediabetes to T2DM [20, 60], however specifics for either treatment have not been identified. Some suggestions include to: reduce overall fat consumption, increase monounsaturated fat intake, increase fiber, and exercise regularly

[17, 193, 209]. While there are numerous drug options for the treatment of T2DM, no medications have been licensed for the treatment of hyperglycemia in conjunction with MetS [17]. Conversely, a variety of studies comparing TLC versus medication for insulin resistance have proven lifestyle intervention to be more successful in greater weight loss, and reducing the incidence of T2DM [17, 33, 210, 211], with further benefit achieved with greater amounts of exercise [93].

Specific dietary protocols for hyperglycemia have been controversial. Grundy et al [28] have found high-monounsaturated fat diets to be more advantageous than high-CHO intakes in regards to insulin resistance, which coincides with the Nurses' Health Study [77] that has found an increased diabetes risk with high glycemic load and trans-fat consumption. Conversely, the San Luis Valley study [29, 212] found no association between CHO intake and hyperinsulinemia. Higher PRO diets have proven beneficial for improving glycemic control [63, 118, 213]. Wycherley et al [42] prefer a combination of high protein and resistance training to reduce insulin concentrations in overweight, type-2 diabetics. Kreider et al [63] have also reported a greater reduction in fasting glucose when performing resistance-based circuit exercise combined with replacing a portion of dietary CHO with PRO. While higher concentrations of protein in the diet may be beneficial, a review of the literature by Dr. Eisenstein et al [115], has found mixed results regarding the effects of a high protein diet (and its effects on glycemic index) on glucose response and insulin sensitivity, and specifically states concern for the use of these diets in patients diagnosed with diabetes.

Aerobic exercise can be beneficial for glycemic control in both single bouts and with persistent training [5], and a range of volumes and intensities will prove efficacious [51]. Chronic exercise may be necessary to sustain the improvements in insulin sensitivity and glucose tolerance, and these benefits may be experienced regardless of age, body weight, and diabetes status [5]. Resistance training is also valuable for increasing the glycogen storage capacity of muscles and thus for long-term glycemic control [5].

Dyslipidemia

Evaluation of the full lipid profile offers two MetS risk factors based on the NCEP ATP III definition of MetS: hypertriglyceridemia ($TG \geq 1.7$ mmol/L) and low HDL (<1.3 mmol/L) [8]. However, if a patient with MetS has an LDL cholesterol above 100 mg/dL (or 2.6 mmol/L), then LDL becomes the primary target for treatment, prior to focusing on reduction of the MetS risk factors specifically [1, 17, 193, 214]. Additionally if a patient with MetS has established CVD or T2DM, the optimal goal for LDL becomes <70 mg/dL (or 1.8 mmol/L) [17, 214].

Poor diet may be to blame for the majority of a dyslipidemic state. Fat consumption, specifically trans- and saturated- fatty acids, is known to increase the concentration of LDL cholesterol and total cholesterol levels [28, 33]. Excessive intake of CHO leads to increased TG concentration, which may play a role in the formation of small LDL particles, and typically also lowers HDL concentrations [28]. Furthermore, the type of CHO consumed may alter the affect on TG; for example, Jenkins [28, 215] found that high-fiber cereal grains did not increase TG.

The state of obesity can further exacerbate dyslipidemia. Excess body weight is significantly related to the rate of daily cholesterol production, causing an increase in TG and an impaired removal of LDL cholesterol [58, 216]. Weight loss has been proven to have a substantial effect on the lipid profile. A meta-analysis has revealed that a one-kilogram decrease in body weight can lead to a decrease in each of the following lipid values: TG (-0.05 mmol/L), LDL cholesterol (-0.02 mmol/L), TG (-0.015 mmol/L), and HDL cholesterol (-0.007 mmol/L) [43, 58]. Additionally, the decrease in HDL cholesterol during weight loss mentioned above has further been shown to increase once weight loss stabilizes [58]. Exercise not only assists with weight reduction, it may independently aid in the normalization of TG and HDL cholesterol levels [33], although other researchers have found that exercise may not impact HDL cholesterol if it is not paired with hypertriglyceridemia at baseline [217].

Dyslipidemia treatment

Similar to each of the other risk factors, a combination of diet and exercise will be most beneficial for reducing the dyslipidemic state. If LDL cholesterol is a primary concern, specific nutritional suggestions include the reduction of cholesterol, saturated- and trans-fat, yet most commonly medication may be prescribed [17]. Once LDL cholesterol is properly managed, the general TLC treatment for atherogenic dyslipidemia may include: weight reduction, caloric restriction with a fat intake between 25-35%, reduced saturated fat and cholesterol consumption, greater intake of omega-3 fatty acids, and a focus on complex CHO and fiber [1, 17, 193]. However, based on the individual's specific lipid profile, specialized treatment may be more beneficial. A higher CHO,

lower fat diet can lead to greater changes in LDL and total cholesterol, while a higher PRO diet may better assist in the lowering of TG and elevation of HDL cholesterol [43]. Yet, other studies have found no effect of protein consumption on blood lipids [115], and the ESNL has found that replacing dietary carbohydrate with a greater proportion of dietary protein can reduce total and LDL cholesterol by 2.5% and 2.8% respectively [83]. Various medications (and combinations) are also prescribed to dyslipidemic individuals [17], with nicotinic acid proving to be quite effective [184], though it is recommended to first attempt TLC for three to six months unless the patient is considered high risk [17].

Additionally, at least 30 minutes of exercise five days a week is advised for individuals with dyslipidemia [193]. Physically active individuals have proven to have lower levels of TG and LDL cholesterol, and higher levels of HDL cholesterol than their sedentary counterparts [5]. A review by Carroll and Dudfield [51] found that even in the absence of weight loss, regular exercise training at a moderate intensity can raise HDL cholesterol and lower TG.

CONCLUSION

MetS is a condition of great concern. Having a combination of three or more risk factors creates an additive effect on disease risk, increasing the risk of both CVD and T2DM. The NCEP ATP III criteria have been proven to be beneficial in identifying MetS and predicting future disease risk. In the US alone, approximately one-quarter of the adult population is diagnosed with MetS. While the prevalence is similar in both men and women, women are reported as having a higher morbidity related to the

disease. Additionally, a large proportion of the US population is sedentary. A TLC program of diet and exercise is the best treatment for preventing and reversing the markers of MetS.

Research from the ESNL has proven that for exercise to effectively improve MetS variables, it should be combined with appropriate dietary modification. However the question remains as to what is “appropriate” dietary modification. The NCEP ATP III promotes a ratio of 15% protein PRO, 50-60% CHO, and 25-30% fat. Conversely, this literature review demonstrated the benefits of reducing CHO consumption, and specifically replacing CHO with higher quantities of PRO, and further benefit with the addition of exercise. Yet, certain health benefits, specifically regarding the markers of MetS, were noted with various macronutrient combinations. Perhaps different dietary recommendations should be prescribed for individuals based on their specific metabolic profile.

CHAPTER III

METHODS

EXPERIMENTAL APPROACH

This study is a retrospective analysis of previous research performed in the Exercise and Sport Nutrition Laboratory (ESNL) that evaluated the effects of higher protein (HP) or higher carbohydrate (HC) diet interventions while participating in a circuit-style resistance-based exercise program. Eight studies were utilized, that recruited overweight and sedentary participants with individual study focuses pertaining to generally healthy, post-menopausal, osteoarthritic, and/or special populations. The demographics of each study are depicted in Table 3.1. Each of these studies initially measured the effectiveness of the Curves® exercise and weight loss program (*Curves International, Waco, TX*) on health outcomes and weight loss in sedentary obese females. This diet and exercise protocol is specifically designed to improve fitness and promote weight loss [218]. For the purposes of this analysis, participants were retrospectively categorized as having $<$ or \geq three risk factors for metabolic syndrome (MetS) utilizing the National Cholesterol Education Program – Adult Treatment Panel III (NCEP ATP III) criteria. Additionally, further analysis was performed categorizing participants based on each metabolic syndrome risk factor independently (high/low waist circumference, high/low triglycerides, low/high HDL cholesterol, high/low blood pressure, and high/low fasting glucose), in order to ascertain how the different dietary protocols affect each risk factor individually.

Table 3.1: Baseline Demographics per Study

Study	N	Age	Height	Weight	BMI	Fat %
1	130	39.1±8.0	164.7±6.6	96.7±19.3	35.6±6.6	44.3±3.2
2	129	38.5±8.1	163.3±6.7	92.6±15.5	34.8±5.7	44.7±4.4
3	135	54.2±4.8	162.8±6.6	91.0±16.9	34.3±6.0	46.4±4.4
4	30	54.3±8.7	163.1±6.9	88.5±13.3	33.3±4.7	46.1±3.1
5	125	49.3±9.7	162.4±6.4	95.6±20.5	36.2±7.1	45.8±4.9
6	46	41.0±11.5	163.8±7.2	88.8±12.2	33.1±4.3	43.9±4.6
7	31	38.3±7.6	162.7±8.2	96.3±19.5	36.2±5.7	45.2±4.3
8	37	65.7±4.9	161.6±5.8	80.2±11.0	30.7±4.0	43.6±3.9
Overall	663	46.2±11.4	163.2±6.7	92.7±17.7	34.8±6.2	45.2±4.3
Height in cm, weight in kg, fat percentage measured via DEXA.						

Participants were prescribed either a HP or HC diet for ten weeks and participated in a supervised circuit-style resistance-training program. Primary outcome measures included the NCEP ATP III risk factors for MetS, which comprise: waist circumference (WC), blood pressure (BP), and fasting levels of triglycerides (TG), HDL cholesterol, and glucose [184]. Secondary outcome measures included body weight, body composition, resting energy expenditure (REE), LDL cholesterol, and total cholesterol. Tertiary outcome measures included markers of cardiovascular and muscular fitness, as well as qualitative measures of psychosocial status. After study completion, participants were retrospectively categorized into two groups, Apparently Healthy (AH) and Metabolic Syndrome (MS), according to their baseline risk level for MetS (using a value of $<$ or \geq three ATP III risk factors respectively). The purpose of this analysis was to determine whether the AH and MS groups were differentially affected by the macronutrient content of the diet (HP vs. HC) combined with the exercise program in regards to the aforementioned outcome measures. Additionally, the effect of the

different diets (HP vs. HC) on each ATP III MetS risk factor was analyzed using the entire study population.

PARTICIPANTS

The research protocols for the studies utilized in this database were reviewed and approved by the Institutional Review Boards at Baylor University and/or Texas A&M University prior to study initiation. The targeted population was sedentary, overweight (BMI >27) females between the ages of 18-75 with no recent participation in a diet or exercise program. Recruitment included referrals from area physicians as well as advertisements posted in local newspapers and television channels, on the Internet, and through campus mail. Interested participants were first pre-screened on the phone to determine eligibility. The following were considered contraindications for participation: 1) presence or diagnosis of any metabolic or cardiovascular disorder including known electrolyte abnormalities (such as heart disease, arrhythmias, diabetes, thyroid disease, or hypogonadism), 2) history of hypertension, hepatorenal, musculoskeletal, autoimmune, or neurological disease, 3) currently taking or prescribed medications for thyroid, hyperlipidemia, hypoglycemia, hypertension, or androgenic medications, 4) having taken ergogenic levels of nutritional supplements that may affect muscle mass (e.g. creatine or HMB), anabolic/catabolic hormone levels (e.g. androstenedione or dehydroepiandrosterone), or weight loss (e.g. ephedra or thermogenics) within three months prior to the start of the study, and/or 5) being pregnant, having been pregnant within the past year, or an interest in becoming pregnant within the next three months. Prior to enrollment in the study, participants with controlled metabolic disorders were

required to obtain medical clearance from their personal physician, affirming that their condition was medically controlled and that it would not influence study results.

Upon passing the telephone pre-screening, eligible participants were then invited to attend a familiarization session to learn more about the study details and complete the necessary paperwork. The familiarization session included the completion of personal and medical history forms, a verbal and written explanation of the study details, and a description of potential risks of participation. Participants were given an opportunity to practice testing procedures and were familiarized with the exercise training equipment. Those participants who still met the eligibility criteria and agreed to the terms of the study were required to sign human subject informed consent statements in compliance with the Human Subjects Guidelines of Baylor University and/or Texas A&M University and the American College of Sports Medicine. A total of 663 women were used in the analysis. Participants were 46 ± 11 years old, 163 ± 7 cm in height, 93 ± 18 kg in weight, and had a BMI of 34.8 ± 6 kg/m² (mean \pm standard deviation).

TESTING SEQUENCE

Table 3.2 shows the general research design and time course for assessments. Participants were tested at baseline (0 weeks), and after ten weeks of performing their assigned exercise and diet intervention. For each testing session, subjects were required to refrain from vigorous physical activity, alcohol intake, and ingestion of over-the-counter medications for 48-hours, as well as maintain a fasted state for 12-hours prior to the appointment. Participants were also required to record all food and fluid intake on dietary record forms for four days (including three week days and one week-end day)

before each testing session. All testing was conducted in the early morning hours, at approximately the same time each day starting at 5:00 am.

Table 3.2: Overview of Research Design and Testing Schedule

Familiarization	Baseline (0 weeks)	10 weeks
Complete Paperwork Review Medical History Sign Informed Consent Dietary Assignment	Diet Record Review Body Weight Waist and Hip Measurements Resting Energy Expenditure BIA ^a DEXA ^b Scan Resting BP ^c and HR ^d Fasting Blood Maximal Cardiopulmonary Exercise Test 1RM ^e and 80% 1RM Isotonic Leg Press and Bench Press Measures Survey Completion ^f	Diet Record Review Body Weight Waist and Hip Measurements Resting Energy Expenditure BIA ^a DEXA ^b Scan Resting BP ^c and HR ^d Fasting Blood Maximal Cardiopulmonary Exercise Test 1RM ^e and 80% 1RM Isotonic Leg Press and Bench Press Measures Survey Completion ^f
^a Bioelectrical Impedance Analysis ^b Dual Energy X-ray Absorptiometry ^c Blood Pressure ^d Heart Rate ^e Repetition Maximum ^f Standardized Quality of Life (SF-36) and Eating Satisfaction Inventory		

During all testing sessions, participants were weighed and had waist- and hip-circumference measured. Resting energy expenditure (REE) was tested using the ParvoMedics TrueMax 2400 Metabolic Measurement System, and body composition was measured using dual-energy x-ray absorptiometry (DEXA). While in a supine rested state, blood pressure and heart rate were measured using standard procedures. Next, approximately 20 mL of fasting blood was then obtained using venipuncture techniques at an antecubital vein. Participants then performed a maximal cardiopulmonary exercise stress test as well as lower and upper body muscular strength

and endurance tests. Additionally, participants completed questionnaires regarding quality of life and body image at each testing session. Participants also completed a weekly medical safety / side effect report that was analyzed by the ESNL research nurse. Subjects were removed from the study if they reported any unusual adverse events in which the supervising nurse or physician recommended discontinuation.

DIETARY INTERVENTION

A carbohydrate/glycemic tolerance questionnaire developed by The Institute for Nutritional Science was used to determine dietary group assignments. Individuals with a positive response on the questionnaire indicated carbohydrate (CHO) intolerance and were assigned to the HP group, whereas the participants with a negative response were assigned to the HC group. The diets were isoenergetic, and low in fat. In order to stimulate weight loss, both the HC and the HP groups were advised to consume 1,200 kcal per day for one week (phase I). Participants were then directed to increase their caloric intake to 1,600 kcal per day for nine weeks (phase II) in order to maintain a steady weight loss without negatively affecting metabolism [219]. Upon initiation of the study, participants were given menus and diet plans to assist with adherence. Participants also discussed their exercise and dietary compliance with a registered dietitian or exercise physiologist every two weeks throughout the ten-week protocol. Overall, participants consumed an average of 1,425 kcal/day. Table 3.3 provides the macronutrient breakdown by percent caloric intake as well as grams per kilogram per day for each dietary group.

Table 3.3: Higher Protein Versus Higher Carbohydrate Dietary Intervention

Diet Group	% Caloric Intake			grams / kg / day		
	PRO	CHO	FAT	PRO	CHO	FAT
HP	29%	35%	36%	1.14	1.41	0.63
HC	18%	51%	31%	0.78	2.20	0.60
HC, higher carbohydrate group; HP, higher protein group; PRO, protein; CHO, carbohydrate						

EXERCISE INTERVENTION

The exercise protocol consisted of three supervised 30-minute circuit-training sessions each week, for the ten-week period (30 workouts total). A trained fitness instructor educated each participant on the proper use of the equipment. Participants performed as many repetitions as possible within thirty seconds on each of the 13 bi-directional machines, with 30-seconds of floor-based calisthenic exercise in between each. Each machine contained calibrated pneumatic resistance pistons that allowed for opposing muscle groups to be trained in a concentric-only fashion. The exercises and machines are listed in Table 3.4. In an interval manner, the calisthenic exercises were utilized to maintain an elevated heart rate corresponding to 60-80% of maximal heart rate (MHR) throughout the workout [220]. Subjects completed two rotations of the circuit, corresponding to exercising for approximately 26 minutes. Participants then cooled down with a standardized whole-body stretching routine. A trained fitness instructor, who monitored exercise intensity and proper technique, supervised each workout. Additionally, workout attendance was recorded to monitor compliance, which was set prior to initiation of the study at a minimum of 70% compliance (21/30 exercise sessions). Although it was not quantified during this study, participants were

additionally encouraged to walk or engage in recreational activities for at least 30 minutes a day on the days that they did not perform circuit training.

Table 3.4: Machines and Exercises Used in Circuit-Style Program

Calisthenic Exercises	Exercise Machines	
running in place	elbow flexion / extension	abdominal crunch / back
high knees	knee flexion / extension	extension
arm circles	shoulder press / lateral pull	pec dec
boxing moves	hip abductor / adductor	oblique twist
stepping	chest press / seated row	shoulder shrug / dip
leg kicks	horizontal leg press	hip extension
	squat	side bends

TESTING PROTOCOLS

Dietary Inventories

Subjects recorded four days (three weekdays and one weekend day) of food and fluid intake prior to each testing session. A registered dietitian analyzed the caloric and macronutrient intakes using ESHA Food Processor (*Version 8.6, 2006, ESHA Research Inc, Salem OR*) Nutritional Analysis software.

Anthropometric Measurements

Height and weight, along with waist and hip circumference measurements were taken at each testing session. Both height and weight were determined utilizing standard procedures on a calibrated electronic scale (*Cardinal Detecto Scale Model 8430, Webb City, Missouri*) with a precision of ± 0.02 kg. Hip and waist circumference were

measured using a tension-controlled tape measure per measurement guidelines established by the American College of Sports Medicine [220].

Resting Energy Expenditure

The ParvoMedics TrueMax 2400 Metabolic Measurement System (*ParvoMedics, Inc., Sandy, UT*) was utilized to assess resting energy expenditure (REE). Fasted subjects rested on an exam table in a supine position with their legs propped up at a 90-degree angle and remained motionless without falling asleep for approximately 20-minutes. A clear metabolic canopy was placed over their head and neck to determine resting oxygen uptake (VO_2) and energy expenditure. Metabolic measurements were taken after the first ten minutes in which the principle variables (such as VO_2 L/min) changed less than 5% within a five-minute period [221]. The manufacturer reported coefficient of variation for this device in lean, healthy individuals is $\pm 2\%$.

Body Composition

Measurements of body composition included total body scanned mass, fat mass, fat free mass, and body fat percentage. Body composition (excluding the cranium) was assessed with the Hologic Discovery W (*Hologic Inc., Waltham, MA*) dual energy x-ray absorptiometer (DEXA) with APEX Software (*APEX Corporation Software, Pittsburg, PA*). Previous research has validated the accuracy of utilizing DEXA for body composition measurement [222, 223]. Test-retest reliability studies on total fat free / soft tissue mass performed on this DEXA machine have previously produced mean coefficients of variation for of 0.31-0.45% with a mean intra-class correlation of 0.985 [224].

Resting Cardiovascular Parameters

Using standard clinical procedures, heart rate and blood pressure were determined in a supine position after resting quietly for five minutes. Heart rate was measured by palpation of the radial artery. A manual mercurial sphygmomanometer (*American Diagnostic Corporation, model #AD-720, Hauppauge, NY*) was utilized for blood pressure measurements, with stethoscope auscultation of the brachial artery [220].

Blood Collection and Analysis

Fasted serum samples were collected using standard phlebotomy techniques via a sterile venipuncture of an antecubital vein. The tubes were immediately centrifuged at 1100 x g for 15 minutes using a standard bench top centrifuge (*Cole Palmer, Vernon Hills, IL, Model # 17250-10*). The serum was then removed with a pipette and placed into micro-centrifuge tubes, frozen at -20°C, and analyzed at a later time for clinical chemistry panels. Serum samples were analyzed for a complete metabolic panel using a calibrated Dade Behring Dimension RXL (*Siemens AG, Munich, Germany*) automated clinical chemistry analyzer. Coefficient of variation for the tests using this analyzer was similar to previously published data for these tests (range: 1.0-9.6%) [225]. In the event that the Dade was not available, serum samples were analyzed by Quest Diagnostics (*Quest Diagnostics, 5850 Rogerdale Road, Houston TX, USA 77072*) using an Olympus AAU 5400 Chemistry Immuno Analyzer (*Olympus America Inc., Center Valley, PA, USA*). Additionally, fasting insulin was assayed in duplicate using a commercially available Enzyme Linked Immunosorbent Assay (ELISA) kit (NO. 80-INSHU-E10, ALPCO, Salem, NH). The BioTek ELX-808 Ultramicroplate reader (BioTek

Instruments Inc, Winooski, VT) was utilized at an optical density of 450 nm against a known standard curve using standard procedures with BioTek Gen5 Analysis software (BioTek Instruments Inc, Winooski, VT). The intra-assay coefficient of variation has been shown to range from 2.9% to 6.2%, with an inter-assay coefficient of variation range of 5.4% to 8.6% (ALPCO, Salem, NH). The Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) was calculated as the product of fasting insulin ($\mu\text{U/mL}$) and fasting glucose (mg/dL) divided by 405 [226].

Fitness Assessments

Maximal Cardiopulmonary Exercise Test

A symptom-limited, Bruce maximal treadmill exercise protocol was performed at each testing session in order to assess peak aerobic capacity (peak VO_2) [220]. The Quinton 710 ECG (*Quinton Instruments, Bothell, WA*), Trackmaster TMX425C treadmill (JAS Fitness Systems, Newton, KS), and Parvo Medics 2400 TrueMax Metabolic Measurement System (*ParvoMedics, Inc., Sandy, UT*) were utilized. The mean coefficient of variation for assessing peak VO_2 with the Bruce protocol has been previously reported to be 6.5% (range of 2.0-14%) [227]. Every morning prior to testing, calibration of gas and flow sensors was completed and found to be within 3% of the previous calibration point.

Throughout the test, HR, BP, and rate of perceived exertion were monitored and heart function was assessed using a standard 12-lead arrangement [220]. Experienced lab assistants conducted all cardiorespiratory treadmill tests. The ECG was reviewed to ensure that no contraindications for exercise testing were present [220]. The Bruce

treadmill protocol was performed following the speeds and grades listed in the standard protocol [228]. An ECG printout, BP, and rate of perceived exertion were obtained near the end of each stage. The participant was encouraged to exercise to their maximum potential unless clinic signs requiring termination of the test became evident [220]. Once the exercise test was complete, the participant performed an active recovery for three minutes, followed by a three-minute seated recovery period.

Isotonic Strength Tests

A standard isotonic Olympic bench press and 45° hip sled / leg press (both from *Nebula Fitness, Versailles, OH*) were utilized to determine upper and lower body maximal strength and muscular endurance. Trained lab assistants experienced in performing exercise testing conducted all strength tests. Hand positioning on the bench press, and seat and foot positioning on the leg press were consistent between testing sessions. Test-retest reliability comparisons for these tests performed by resistance-trained subjects in the ESNL have produced low mean coefficients of variation and high reliability (bench press 1.9%, intraclass $r=0.94$ and leg press: 0.7%, intraclass $r=0.91$) [229].

To assess both upper and lower body strength, participants performed a one rep maximum (1RM) protocol. Each individual started with a warm-up (two sets of ten repetitions at approximately 50% of their anticipated 1RM). Next participants experienced a progressive increase (five to ten lbs. on bench press, 10-25 lbs on leg press) where they attempted a single-repetition, and rested two minutes before attempting another increase, until 1RM on the bench press was achieved. Following

determination of the participant's 1RM, subjects rested for four minutes and then performed an upper body muscular endurance test by performing as many repetitions as possible without stopping, at a calculated weight of 80% of their 1RM [230].

Psychosocial Assessments

In order to measure the psychosocial dimensions that may change due to the experience-of and results-gained throughout the study, participants completed the SF-36 Health-Related Quality of Life and also the Body Image Questionnaire at each testing session (See Appendices G and H) [231, 232]. The SF-36 assessed various physical and mental components to include: physical functioning (the ability to perform most vigorous physical activities without limitation to health), role physical (the ability to work and perform daily activities), bodily pain (limitations due to pain), general health (assessment of personal health), vitality (perception of energy level), social functioning (ability to perform normal social activities), role emotion (problems with work or other daily activities), and mental health (state of feelings of peacefulness, happiness, and calmness). The Body Image Questionnaire is a compilation of three questionnaires: the Rosenberg Self-Esteem Scale (RSE), which assesses unidimensional global self-esteem, the Social Physique Anxiety Scale (SPAS), and the Multidimensional Body-Self Relations Questionnaire (MBSRQ-AS), which assesses self-attitudinal disposition towards the physical construct including appearance evaluation, appearance orientation, overweight preoccupation, self-classified weight, and body areas satisfaction scale [233-235].

STATISTICAL ANALYSIS

All subjects who completed the studies were included in the analysis. The series mean method was used to replace any missing data points with the exception of waist circumference. One study utilized in this analysis did not record waist circumference. Therefore, a one-way analysis of variance (ANOVA) was used to determine the average weight and body fat percentage for individuals that met the MetS criteria for elevated waist circumference. All participants missing this data who met this criteria were coded with a “1” for having the MetS criteria. The mean value of all of the participants coded “0” or “1” for not having or having the risk factor respectively was calculated, and used as a replacement value for the missing waist circumference measurements.

The analysis was performed retrospectively on 663 women (n=663) from eight previous weight loss studies in the ESNL. Participants were retrospectively categorized into two groups, Apparently Healthy (AH) or Metabolic Syndrome (MS), based on the presence of MetS as identified using the NCEP ATP III criteria ($<$ or \geq three risk factors, respectively). A secondary analysis was conducted to determine the effect of HP and HC diets with each MetS risk factor independently stratified for the entire study population.

The statistical analysis was performed using SPSS (*Version 20, IBM Corporation, Armonk, NY*). Participant baseline demographic data were analyzed by ANOVA. Related variables were grouped together and analyzed by multivariate analysis of variance (MANOVA) with repeated measures. Overall MANOVA effects were examined using the Wilks' Lambda time, time x diet, time x MS, and time x diet x

MS p-levels. Greenhouse-Geisser univariate tests of within-subjects time, time x diet, time x MS, and time x diet x MS effects and between-subjects univariate group effects were reported for each variable analyzed within the MANOVA model. Non-correlated variables were analyzed by repeated measures ANOVA with univariate diet, MS, time, time x diet, time x MS, and time x diet x MS interaction effects reported. Delta values or percent difference were calculated and analyzed on select variables by ANOVA for repeated measures to assess the changes and normalize any differences in baseline values. Delta values were calculated by subtracting the baseline testing session (T1) from ten-week testing session (T2-T1). Percent differences were calculated by subtracting T1 from T2, dividing by T1, then multiplying by 100 $[(T2-T1)/T1 \cdot 100]$.

Data were considered statistically significant when the probability of type I error was ≤ 0.05 , and statistical trends were considered when the probability of error ranged between $p > 0.05$ to $p \leq 0.10$. Tukey's least significant difference post hoc analysis was performed to determine where significance between group-combinations was obtained. Unless otherwise stated, data are presented throughout the text as mean \pm standard deviation ($X \pm SD$). The sample had sufficient power (HC: $n=291$; HP: $n=370$; need $n=217$ per group for a power of 0.8 at the 0.05 α -level) to assess statistically significant changes in outcome measures.

CHAPTER IV

RESULTS

BASELINE DEMOGRAPHICS

Table 4.1 presents the baseline demographics for each of the diet and health status groups. Data from a total of 663 participants were used in the analysis.

Participants were 46.2 ± 11.4 years old, 163 ± 7 cm in height, 92.7 ± 18 kg in weight, and categorized as obese with a BMI of 34.8 ± 6 kg/m² (mean \pm standard deviation).

Significant baseline differences were observed between the diet groups for weight (HP 96 ± 19 , HC 89 ± 15 kg, $p < 0.001$), body mass index (HP 36 ± 7 , HC 33 ± 5 kg/m², $p < 0.001$) and DEXA fat percentage (HP 46 ± 4 , HC $45 \pm 4\%$, $p = 0.023$). Additionally, significant baseline differences were observed between the health status groups for age (AH 45 ± 12 , MS 48 ± 11 years, $p = 0.013$), weight (AH 90 ± 18 , MS 96 ± 18 kg, $p < 0.001$), and body mass index (AH 34 ± 6 , MS 36 ± 6 kg/m², $p < 0.001$).

The numerical and percent frequencies of each metabolic syndrome risk factor are listed in Table 4.2. In order of prevalence, waist circumference (80%) was the most prevalent risk factor for this study population, which was to be expected due to recruitment of overweight participants. Next, glucose (41%) and HDL cholesterol (40%) demonstrated similar levels of prevalence, then blood pressure (37%), and finally elevated triglycerides were the least frequent risk factor observed (33%). Table 4.3 represents the N-sizes for the number of risk factors per diet group. Overall, 43% of the participants (N=286) had three or more risk factors and were characterized as having metabolic syndrome.

Table 4.1: Baseline Demographics for All Diet and Health Status Groups

Variable	Group	Baseline		P-level
Age (Years)	HP-AH	44.6	± 11.3	D = 0.17
	HP-MS	47.1	± 10.6	M = 0.013
	HC-AH	46.1	± 12.3	D x M = 0.82
	HC-MS	48.1	± 11.0	
	HP	45.8	± 11.0	
	HC	46.9	± 11.8	
	AH	45.3	± 11.8	
	MS	47.5 [‡]	± 10.7	
	All	46.2	± 11.4	
Height (cm)	HP-AH	163.5	± 6.6	D = 0.74
	HP-MS	162.7	± 6.8	M = 0.69
	HC-AH	163.1	± 6.5	D x M = 0.24
	HC-MS	163.5	± 7.1	
	HP	163.2	± 6.7	
	HC	163.3	± 6.7	
	AH	163.3	± 6.6	
	MS	163.0	± 6.9	
	All	163.2	± 6.7	
Weight (kg)	HP-AH	93.7	± 19.0	D = 0.001
	HP-MS	98.1	± 18.9	M = 0.001
	HC-AH	86.5	± 14.8	D x M = 0.60
	HC-MS	92.3	± 14.4	
	HP	95.8 [†]	± 19.1	
	HC	88.8	± 14.9	
	AH	90.3	± 17.5	
	MS	95.8 [‡]	± 17.5	
	All	92.7	± 17.7	
Body Mass Index (kg/m ²)	HP-AH	35.0	± 6.6	D = 0.001
	HP-MS	37.0	± 6.4	M = 0.001
	HC-AH	32.5	± 5.3	D x M = 0.99
	HC-MS	34.5	± 4.9	
	HP	35.9 [†]	± 6.6	
	HC	33.3	± 5.2	
	AH	33.8	± 6.1	
	MS	36.0 [‡]	± 6.0	
	All	34.8	± 6.2	
DEXA Body Fat (%)	HP-AH	45.5	± 4.3	D = 0.023
	HP-MS	45.5	± 4.5	M = 0.80
	HC-AH	44.7	± 4.4	D x M = 0.85
	HC-MS	44.8	± 3.8	
	HP	45.5 [†]	± 4.4	
	HC	44.7	± 4.2	
	AH	45.1	± 4.4	
	MS	45.2	± 4.3	
	All	45.2	± 4.3	
<p>HP = higher protein, HC = higher carbohydrate, AH = apparently healthy, MS = metabolic syndrome risk factor, D = diet effect, M = metabolic syndrome risk factor effect, D x M = diet by metabolic syndrome risk factor effect. Values are means ± standard deviations from 198 participants in the HP-AH group, 173 in the HP-MS group, 179 in the HC-AH group, 113 in the HC-MS group, 371 in the HP group, 292 in the HC group, 377 in the AH group, 286 in the MS group, and 663 participants total. [†] Significant diet effect, p < 0.05. [‡] Significant metabolic syndrome effect, p < 0.05.</p>				

Overall, 43% of the participants (N=286) had three or more risk factors and were characterized as having metabolic syndrome.

Table 4.2: Baseline Frequencies per Metabolic Syndrome Risk Factor

Risk Factor	Baseline		
	HP	HC	TTL
Waist Circumference	316 (85%)	215 (74%)	531 (80%)
Glucose	157 (42%)	116 (40%)	273 (41%)
HDL Cholesterol	151 (41%)	112 (38%)	263 (40%)
Blood Pressure	148 (40%)	96 (33%)	244 (37%)
Triglycerides	124 (33%)	96 (33%)	220 (33%)
HP, higher protein; HC, higher carbohydrate; TTL, total N-size. Percentages are based on the N-size for HP (N=371), HC (N=292), and total (N=663)			

Table 4.3: Number of Risk Factors per Diet Group

Number of Risk Factors	Baseline			
	HP	HC	Total	
0	18	26	44	Apparently Healthy 57%
1	69	61	130	
2	111	92	203	
3	105	68	173	Metabolic Syndrome 43%
4	50	39	89	
5	18	6	24	
HP, higher protein (N=371); HC, higher carbohydrate (N=292), Total (N=663).				

ANALYSIS OF ENERGY INTAKE

An analysis utilizing one-way ANOVA on the baseline values for each of the nutrition variables (expressed in grams/day, grams/kg/day, and percent intake) revealed no significant differences between the diet groups (see baseline values on tables 4.4-4.6), with the exception of protein reported in g/d ($p<0.001$). However, when expressed relative to

body weight (g/kg/d), the baseline values for protein were not significantly different between the HP and HC diet groups ($p=0.07$). Table 4.4 depicts the time x diet MANOVA for total energy intake in kcal/day as well as macronutrient intake in g/day. The MANOVA revealed an overall time (Wilks' Lambda $p<0.001$) and time x diet (Wilks' Lambda $p<0.001$) effect. Dieting reduced energy intake in both groups (-452 ± 616 kcal/day), with a significantly greater decrease reported in the HP group (HP -507 ± 624 , HC -383 ± 599 kcal/day, $p=0.01$). Throughout the study, protein intake significantly increased in the HP group ($+22.1\pm42$ g/day) and decreased in the HC group (-6.4 ± 24.7 g/day, $p<0.001$). While carbohydrate intake decreased in both groups during the study protocol, a significantly greater decrease was reported for the higher protein group (HP -99.3 ± 90.5 , HC -36.1 ± 79.9 g/day, $p<0.001$). Post-hoc analysis revealed that participants in the HP group consumed more protein (HP 100 ± 36 , HC 66 ± 20 g/day, $p<0.001$) and less carbohydrate (HP 125 ± 55 , HC 184 ± 48 g/day, $p<0.001$) than those in the HC group. Fat intake decreased in similar amounts for each group throughout the study protocol (-22.7 ± 32.1 g/day), while the higher protein group consumed significantly more fat (in grams/day) throughout the study (HP 66.7 ± 0.9 , HC 62.4 ± 1.0 g/day, mean (SEM), $p=0.001$).

Table 4.4: Dietary Intake Before and After Ten Weeks of Exercise and Dietary Intervention Expressed in Grams

Variable	Group	Baseline		10 Weeks		Group (SEM)		P-level
Energy Intake (kcal/day)	HP	1,908	± 498	1,402 ^b	± 377	1,655	± 17	T = 0.001
	HC	1,835	± 575	1,452 ^b	± 323	1,644	± 19	D = 0.66
	Time	1,876	± 534	1,424 [*]	± 355			T x D = 0.01
Protein Intake (g/day)	HP	78.0	± 20.7	100.1 ^b	± 36.1	89.0 [†]	± 0.9	T = 0.001
	HC	72.2 ^a	± 18.7	65.8 ^{ab}	± 19.8	69.0	± 1.1	D = 0.001
	Time	75.4	± 20.1	85.0 [*]	± 34.5			T x D = 0.001
Carbohydrate Intake (g/day)	HP	224.3	± 76.8	125.0 ^b	± 54.7	174.6 [†]	± 2.6	T = 0.001
	HC	220.6	± 78.8	183.5 ^{ab}	± 48.4	201.5	± 2.9	D = 0.001
	Time	222.2	± 77.6	150.7 [*]	± 59.6			T x D = 0.001
Fat Intake (g/day)	HP	77.7	± 24.5	55.8 ^b	± 20.9	66.7 [†]	± 0.9	T = 0.001
	HC	74.2	± 30.8	50.6 ^{ab}	± 16.3	62.4	± 1.0	D = 0.001
	Time	76.2	± 27.5	53.5 [*]	± 19.2			T x D = 0.51
HP = higher protein, HC = higher carbohydrate, T = time effect, D = diet effect, T x D = time by diet effect. Values are means ± standard deviations (except group means are means ± standard error) from 371 in the HP group, 292 in the HC group, and 663 participants total. [*] Significantly different than baseline, p < 0.05 (univariate). [†] Significant diet effect, p < 0.05 (univariate). ^a Significantly different than HP group, P < 0.05 (post hoc LSD). ^b Significantly different than baseline, p < 0.05 (post hoc LSD).								

Table 4.5: Dietary Intake Before and After Ten Weeks of Exercise and Dietary Intervention Expressed in Grams per Kilogram

Variable	Group	Baseline		10 Weeks		Group (SEM)		P-level
Energy Intake (kcal/kg/day)	HP	20.4	± 5.8	15.9 ^b	± 5.2	18.2 [†]	± 0.2	T = 0.001
	HC	21.0	± 6.6	17.4 ^{ab}	± 4.7	19.2	± 0.3	D = 0.003
	Time	20.7	± 6.1	16.6 [*]	± 5.0			T x D = 0.09
Protein Intake (g/kg/day)	HP	0.83	± 0.2	1.14 ^b	± 0.5	0.99 [†]	± 0.01	T = 0.001
	HC	0.83	± 0.2	0.78 ^a	± 0.3	0.81	± 0.02	D = 0.001
	Time	0.83	± 0.2	0.98 [*]	± 0.4			T x D = 0.001
Carbohydrate Intake (g/kg/day)	HP	2.40	± 0.9	1.41 ^b	± 0.7	1.91 [†]	± 0.03	T = 0.001
	HC	2.52	± 0.9	2.20 ^{ab}	± 0.7	2.36	± 0.04	D = 0.001
	Time	2.46	± 0.9	1.76 [*]	± 0.8			T x D = 0.001
Fat Intake (g/kg/day)	HP	0.83	± 0.3	0.63 ^b	± 0.3	0.73	± 0.01	T = 0.001
	HC	0.85	± 0.3	0.60 ^b	± 0.2	0.73	± 0.01	D = 0.79
	Time	0.84	± 0.3	0.62 [*]	± 0.2			T x D = 0.10
HP = higher protein, HC = higher carbohydrate, T = time effect, D = diet effect, T x D = time by diet effect. Values are means ± standard deviations (except group means are means ± standard error) from 371 in the HP group, 292 in the HC group, and 663 participants total. [*] Significantly different than baseline, p < 0.05 (univariate). [†] Significant diet effect, p < 0.05 (univariate). ^a Significantly different than HP group, P < 0.05 (post hoc LSD). ^b Significantly different than baseline, p < 0.05 (post hoc LSD).								

Table 4.6: Dietary Intake Before and After Ten Weeks of Exercise and Dietary Intervention Expressed in Percentages

Variable	Group	Baseline		10 Weeks		Group (SEM)		P-level
Energy Intake (kcal//day)	HP	1,908	± 498	1,402 [*]	± 377	1,655	± 17	T = 0.001
	HC	1,835	± 575	1,452 [*]	± 323	1,644	± 19	D = 0.66
	Time	1,876	± 534	1,424 [*]	± 355			T x D = 0.010
Protein Intake (%)	HP	17.1	± 6.2	29.2 [*]	± 8.7	23.1 [†]	± 0.3	T = 0.001
	HC	16.3	± 3.9	18.4 ^{ab}	± 4.7	17.3	± 0.3	D = 0.001
	Time	16.7	± 5.3	24.4 [*]	± 9.0			T x D = 0.001
Carbohydrate Intake (%)	HP	46.5	± 8.1	35.3 [*]	± 9.9	40.9 [†]	± 0.3	T = 0.001
	HC	47.6	± 7.6	50.5 ^{ab}	± 7.1	49.1	± 0.4	D = 0.001
	Time	47.0	± 7.9	42.0 [*]	± 11.5			T x D = 0.001
Fat Intake (%)	HP	36.4	± 5.8	35.5 [*]	± 7.3	36.0 [†]	± 0.3	T = 0.001
	HC	36.1	± 6.4	31.2 ^{ab}	± 6.1	33.6	± 0.3	D = 0.001
	Time	36.3	± 6.1	33.6 [*]	± 7.1			T x D = 0.001
HP = higher protein, HC = higher carbohydrate, T = time effect, D = diet effect, T x D = time by diet effect. Values are means ± standard deviations (except group means are means ± standard error) from 371 in the HP group, 292 in the HC group, and 663 participants total. * Significantly different than baseline, p < 0.05 (univariate). † Significant diet effect, p < 0.05 (univariate). ^a Significantly different than HP group, P < 0.05 (post hoc LSD). ^b Significantly different than baseline, p < 0.05 (post hoc LSD).								

As Tables 4.5 and 4.6 present, similar findings were observed when energy intake data were expressed in kcals/kg/day and macronutrient percentage of daily energy intake. Yet, Table 4.5 also demonstrates that relative to body weight, a group effect was observed for total energy intake, demonstrating the higher carbohydrate group consumed more calories throughout the study (HP 18.2±0.2, HC 19.2±0.3 kcal/kg/d, mean (SEM), p=0.003), and those in the HP group tended to experience greater decreases in total energy consumption (HP -4.5±6.7, HC -3.6±6.5 kcal/kg/d, p=0.09). Additionally, when expressed as g/kg/day (Table 4.5), participants in the higher carbohydrate group tended to experience a greater decrease in fat intake (HP -0.20±0.33, HC -0.24±0.35 g/kg/d, p=0.10). This relationship was found to be significant when expressed as percent of macronutrient consumption, with the HC group experiencing a significantly greater decrease in fat intake (HP -1.0±8.4, HC -4.9±8.1%, p<0.001). Figures 4.1 and 4.2 depict

the relative macronutrient consumption (g/kg/d) for protein, carbohydrate, and fat at baseline and ten-weeks, and reveal the significantly greater protein consumption in the HP group and carbohydrate consumption in the HC group. Hypothesis H₁ was therefore accepted in that statistically significant difference were observed between the HP and HC groups regarding macronutrient intake.

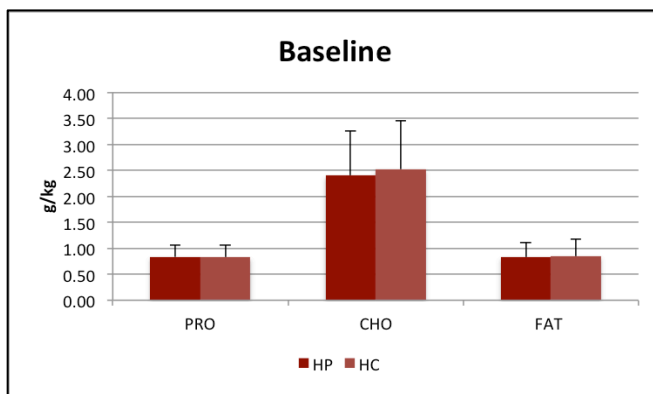


Figure 4.1: Comparison of Macronutrient Consumption at Baseline

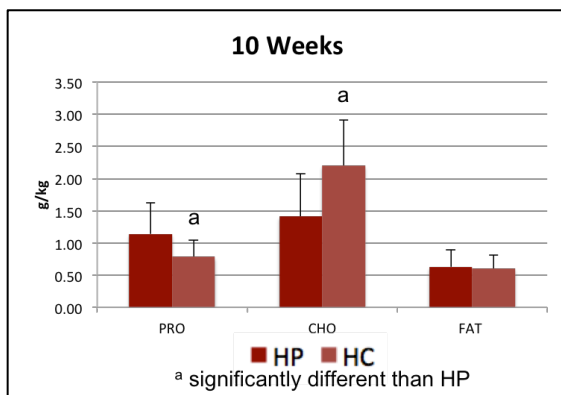


Figure 4.2: Comparison of Macronutrient Consumption at Ten Weeks

ANALYSIS OF METABOLIC SYNDROME STATUS

Table 4.7 depicts the time x metabolic syndrome risk factor MANOVA for the metabolic syndrome risk factors. A baseline ANOVA of the NCEP ATP III metabolic syndrome risk factor variables confirmed that each risk factor was significantly different between the groups (apparently healthy vs. metabolic syndrome) at baseline. This was to be expected, considering the groups were stratified based on whether or not they expressed these metabolic syndrome risk factors. As anticipated, the MANOVA revealed an overall time (Wilks' Lambda $p<0.001$) and time x metabolic syndrome risk factor (Wilks' Lambda $p<0.001$) effect for the metabolic syndrome variables. In regards to health status, both groups (AH and MS) significantly reduced the mean values for each risk factor over the course of the study (time effect, $p<0.001$), with a significantly greater decrease found in the MS group for all variables (time x metabolic syndrome effect, $p<0.001$) except waist circumference ($p=0.12$) and HDL cholesterol (which experienced a significantly greater decrease in the AH group, $p=0.011$).

Analysis of MANOVA univariate tests revealed significant time x metabolic syndrome risk factor effects in which the MS group demonstrated a significantly greater decrease in systolic blood pressure (AH -0.47 ± 13.3 , MS -5.91 ± 16.0 mmHg, $p<0.001$), diastolic blood pressure (AH -0.35 ± 8.92 , MS -4.08 ± 10.5 mmHg, $p<0.001$), triglycerides (AH -0.00 ± 0.47 , MS -0.23 ± 0.73 mmol/L, $p<0.001$), and glucose (AH $+0.01\pm0.73$, MS -0.24 ± 1.19 mmol/L, $p=0.001$) than the AH group.

Table 4.7: Changes in Metabolic Syndrome Risk Factor Values After Ten Weeks of Exercise and Dietary Intervention

Variable	Group	Baseline		10 Weeks		Group (SEM)		P-level
Waist Circumference (cm)	AH	95.9	± 12.5	92.5 [*]	± 12.4	94.2	± 0.6	T = 0.001
	MS	102.9 ^b	± 12.6	98.8 ^{bs}	± 11.9	100.8 [‡]	± 0.7	M = 0.001
	Time	98.9	± 13.0	95.2 [*]	± 12.6			T x M = 0.12
Systolic Blood Pressure (mmHg)	AH	120.9	± 12.2	120.5	± 13.2	120.7	± 0.6	T = 0.001
	MS	130.4 ^b	± 15.7	124.4 ^{bs}	± 14.6	127.4 [‡]	± 0.7	M = 0.001
	Time	125.0	± 14.6	122.2 [*]	± 13.9			T x M = 0.001
Diastolic Blood Pressure (mmHg)	AH	78.0	± 8.4	77.6	± 9.0	77.8	± 0.4	T = 0.001
	MS	83.6 ^b	± 9.6	79.5 ^{bs}	± 9.0	81.6 [‡]	± 0.4	M = 0.001
	Time	80.4	± 9.4	78.4 [*]	± 9.1			T x M = 0.001
Triglycerides (mmol/L)	AH	1.22	± 0.5	1.22	± 0.6	1.22	± 0.03	T = 0.001
	MS	2.01 ^b	± 0.9	1.78 ^{bs}	± 0.9	1.89 [‡]	± 0.04	M = 0.001
	Time	1.56	± 0.8	1.46 [*]	± 0.8			T x M = 0.001
HDL Cholesterol (mmol/L)	AH	1.49	± 0.3	1.41 [*]	± 0.3	1.45	± 0.01	T = 0.001
	MS	1.24 ^b	± 0.3	1.20 ^{bs}	± 0.3	1.22 [‡]	± 0.02	M = 0.001
	Time	1.39	± 0.3	1.32 [*]	± 0.3			T x M = 0.011
Glucose (mmol/L)	AH	5.30	± 0.8	5.30	± 0.7	5.30	± 0.05	T = 0.002
	MS	6.15 ^b	± 1.6	5.91 ^{bs}	± 1.4	6.03 [‡]	± 0.06	M = 0.001
	Time	5.66	± 1.3	5.57 [*]	± 1.1			T x M = 0.001
AH = apparently healthy, MS = metabolic syndrome risk factor group, T = time effect, M = metabolic syndrome risk factor effect, T x M = time by metabolic syndrome risk factor effect. Values are means ± standard deviations (except group means are means ± standard error) from 377 in the AH group, 286 in the MS group, and 663 participants total. [*] Significantly different than baseline, p < 0.05 (univariate). [‡] Significant metabolic syndrome effect, p < 0.05 (univariate). ^b Significantly different than AH group, p < 0.05 (post hoc LSD). ^s Significantly different than baseline, p < 0.05 (post hoc LSD).								

However, the AH group experienced a significantly greater reduction in HDL cholesterol (AH -0.09±0.25, MS -0.04±0.21 mmol/L, p=0.011). While overall waist circumference significantly decreased over time (-3.65±5.7 cm), no significant differences were seen between the AH and MS groups in centimeters lost throughout the study (p=0.12). Comparison of the delta change from baseline for each metabolic syndrome risk factor can be seen in Figures 4.3 and 4.4. Hypothesis H₂ was therefore accepted in that statistically significant differences were observed between the AH and MS groups in the markers of metabolic syndrome.

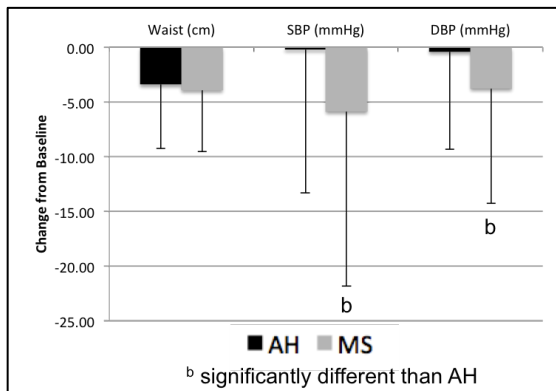


Figure 4.3: Delta Change in Metabolic Syndrome Risk Factors Over the Ten Week Study; Waist Circumference and Blood Pressure

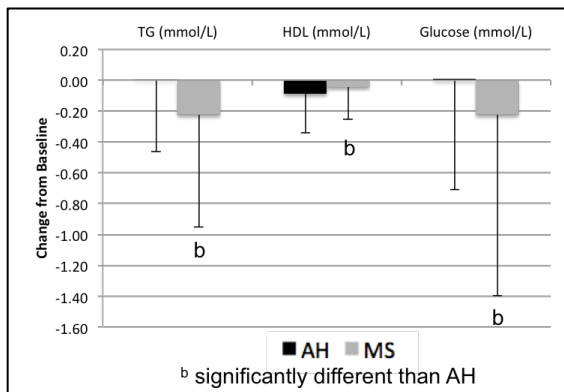


Figure 4.4: Delta Change in Metabolic Syndrome Risk Factors Over the Ten Week Study; Triglycerides, HDL, and Glucose

ANALYSIS OF METABOLIC SYNDROME AS A RISK FACTOR

Three-way MANOVAs and/or ANOVAs have been utilized to analyze all variables measured on participants regarding time x diet x metabolic syndrome risk factor status. The next 11 tables represent the mean values and p-levels for each analysis regarding the metabolic syndrome risk factor.

Energy Intake

Table 4.8 depicts the time x diet x metabolic syndrome risk factor MANOVA for total energy intake in kcals/kg/day as well as macronutrient intake in g/kg/day. The MANOVA revealed an overall time (Wilks' Lambda $p < 0.001$) and time x diet (Wilks' Lambda $p < 0.001$) effect, but no significant time x metabolic syndrome risk factor (Wilks' Lambda $p = 0.24$) or time x diet x metabolic syndrome risk factor (Wilks' Lambda $p = 0.20$) interactions. Post hoc analysis on the ten-week values was utilized to determine the following: Participants in the HC group and the AH group consumed significantly more calories relative to body weight (HP 15.9 ± 5.2 , HC 17.4 ± 4.7 kcal/kg/d, $p < 0.001$) (AH 17.0 ± 5.4 , MS 16.0 ± 4.4 kcal/kg/d, $p = 0.007$). Further review of the individual group-combinations revealed that total energy consumption was significantly different in each group, with the HC-AH group consuming significantly more energy than the other groups (17.9 ± 5.1 kcal/kg/d, $p = 0.033$). Participants in the HP group and the AH group consumed significantly more protein relative to body weight (HP 1.14 ± 0.5 , HC 0.78 ± 0.3 g/kg/d, $p < 0.001$) (AH 1.01 ± 0.5 , MS 0.96 ± 0.4 g/kg/d, $p = 0.013$). Specifically, the HP-AH group consumed significantly more protein than HC-AH ($p < 0.001$), and HP-MS consumed significantly more protein than HC-MS ($p < 0.001$). The HC group consumed significantly more carbohydrate relative to body weight (HP 1.41 ± 0.7 , HC 2.20 ± 0.7 g/kg/d, $p < 0.001$), with no significant difference observed between the risk factor groups ($p = 0.09$).

Table 4.8: Dietary Intake Before and After Ten Weeks of Exercise and Dietary Intervention Based on Metabolic Syndrome Risk Factor Status

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Energy Intake (kcal/kg/day)	HP-AH	20.52	± 5.64	16.29 ^g	± 5.62				T = 0.001
	HP-MS	20.28	± 5.92	15.45 ^g	± 4.55				D = 0.007
	HC-AH	21.35	± 6.80	17.89 ^{e,g}	± 5.05				M = 0.028
	HC-MS	20.55	± 6.21	16.62 ^{d,f,g}	± 4.03				D x M = 0.49
	HP	20.41	± 5.76	15.90 ^g	± 5.16	18.14 [†]	± 0.23		T x D = 0.11
	HC	21.04	± 6.58	17.39 ^{ag}	± 4.71	19.10	± 0.27		T x M = 0.31
	AH	20.91	± 6.22	17.05 ^g	± 5.41	19.01	± 0.23		T x D x M = 0.90
	MS	20.39	± 6.03	15.91 ^{bg}	± 4.38	18.23 [‡]	± 0.27		
	Time	20.69	± 6.14	16.56 [*]	± 5.02				
Protein Intake (g/kg/day)	HP-AH	0.85	± 0.25	1.18 ^g	± 0.55				T = 0.001
	HP-MS	0.82	± 0.21	1.10 ^g	± 0.40				D = 0.001
	HC-AH	0.84	± 0.23	0.82 ^c	± 0.28				M = 0.006
	HC-MS	0.80	± 0.22	0.73 ^d	± 0.20				D x M = 0.74
	HP	0.83	± 0.23	1.14 ^g	± 0.49	0.99 [†]	± 0.01		T x D = 0.001
	HC	0.83	± 0.23	0.78 ^a	± 0.26	0.80	± 0.02		T x M = 0.15
	AH	0.84	± 0.24	1.01 ^g	± 0.48	0.92	± 0.01		T x D x M = 0.93
	MS	0.81	± 0.22	0.96 ^{bg}	± 0.38	0.87 [‡]	± 0.02		
	Time	0.83	± 0.23	0.98 [*]	± 0.44				
Carbohydrate Intake (g/kg/day)	HP-AH	2.40	± 0.86	1.42 ^g	± 0.69				T = 0.001
	HP-MS	2.40	± 0.87	1.41 ^g	± 0.62				D = 0.001
	HC-AH	2.54	± 0.95	2.27 ^{c,g}	± 0.76				M = 0.28
	HC-MS	2.50	± 0.91	2.10 ^{d,f,g}	± 0.61				D x M = 0.32
	HP	2.40	± 0.87	1.41 ^g	± 0.66	1.91 [†]	± 0.03		T x D = 0.001
	HC	2.52	± 0.93	2.20 ^{ag}	± 0.71	2.35	± 0.04		T x M = 0.31
	AH	2.47	± 0.91	1.82 ^g	± 0.84	2.16	± 0.03		T x D x M = 0.42
	MS	2.44	± 0.89	1.68 ^g	± 0.70	2.10	± 0.04		
	Time	2.46	± 0.90	1.76 [*]	± 0.79				
Fat Intake (g/kg/day)	HP-AH	0.84	± 0.28	0.66 ^g	± 0.29				T = 0.001
	HP-MS	0.82 ^c	± 0.27	0.60 ^g	± 0.22				D = 0.65
	HC-AH	0.87	± 0.34	0.61 ^g	± 0.23				M = 0.022
	HC-MS	0.81	± 0.31	0.59 ^g	± 0.19				D x M = 0.93
	HP	0.83	± 0.27	0.63 ^g	± 0.26	0.73	± 0.01		T x D = 0.15
	HC	0.85	± 0.33	0.60 ^g	± 0.21	0.72	± 0.01		T x M = 0.86
	AH	0.85	± 0.31	0.64 ^g	± 0.26	0.75	± 0.01		T x D x M = 0.23
	MS	0.82	± 0.28	0.60 ^{bg}	± 0.21	0.71 [‡]	± 0.01		
	Time	0.84	± 0.30	0.62 [*]	± 0.24				

HP = higher protein, HC = higher carbohydrate, AH = apparently healthy, MS = metabolic syndrome risk factor, T = time effect, D = diet effect, M = metabolic syndrome risk factor effect, D x M = diet by metabolic syndrome risk factor effect, T x D = time by diet effect, T x M = time by metabolic syndrome risk factor effect, T x D x M = time by diet by metabolic syndrome risk factor effect.

Values are means ± standard deviations (except group means are means ± standard error) from 198 participants in the HP-AH group, 173 in the HP-MS group, 179 in the HC-AH group, 113 in the HC-MS group, 371 in the HP total group, 292 in the HC total group, 377 in the AH group, 286 in the MS group, and 663 participants total.

* Significantly different than baseline, $p < 0.05$ (univariate). † Significant diet effect, $p < 0.05$ (univariate). ‡ Significant metabolic syndrome effect, $p < 0.05$ (univariate). ^a Significantly different than HP group, $p < 0.05$ (post hoc LSD). ^b Significantly different than AH group, $p < 0.05$ (post hoc LSD). ^c Significantly different than HP-AH group, $p < 0.05$ (post hoc LSD). ^d Significantly different than HP-MS group, $p < 0.05$ (post hoc LSD). ^e Significantly different than HC-AH group, $p < 0.05$ (post hoc LSD). ^f Significantly different than baseline, $p < 0.05$ (post hoc LSD).

The HC-AH group consumed significantly more carbohydrate than HP-AH ($p<0.001$) and HC-MS ($p=0.033$), and HC-MS consumed significantly more carbohydrate than HP-MS ($p<0.001$). Finally, participants categorized as apparently healthy consumed significantly more fat (AH 0.64 ± 0.3 , MS 0.60 ± 0.2 g/kg/d, $p=0.034$), with the HP-AH group consuming a significantly greater amount than HP-MS ($p=0.027$).

Body Composition

The variables related to body composition are broken down into two tables of analysis: DEXA scan measurements and anthropometric measurements. All of the variables within the DEXA measurements were analyzed using a three-way MANOVA, while the anthropometric variables were each analyzed using individual three-way ANOVAs.

Table 4.9 depicts the time x diet x metabolic syndrome risk factor MANOVA for DEXA scan measurements. The three-way MANOVA revealed an overall time (Wilks' Lambda $p<0.001$) and time x diet (Wilks' Lambda $p=0.036$) effect for the DEXA variables, but no significant time x metabolic syndrome risk factor (Wilks' Lambda $p=0.35$) or time x diet x metabolic syndrome risk factor (Wilks' Lambda $p=0.52$) effect. However, there was a significant time x metabolic syndrome risk factor interaction (Wilks' Lambda $p=0.010$) when run as a two-way MANOVA. Analysis of MANOVA univariate tests revealed significant time effects ($p<0.001$) in changes in total scanned mass (-3.5 ± 3.5 kg), fat mass (-2.8 ± 2.8 kg), lean mass (-0.7 ± 2.0 kg), and fat percentage ($-1.5\pm1.9\%$).

Table 4.9: Body Composition Before and After Ten Weeks of Exercise and Dietary Intervention Based on Metabolic Syndrome Status and Measured Via DEXA Scan

Variable	Group	Baseline		10 Weeks			Group (SEM)			P-level
Scanned Mass (kg)	HP-AH	87.3	± 18.0	83.6 [§]	± 17.6				T = 0.001	
	HP-MS	91.5 ^c	± 17.9	87.3 ^{c,§}	± 17.1				D = 0.001	
	HC-AH	80.3 ^c	± 14.3	77.5 ^{c,§}	± 13.6				M = 0.001	
	HC-MS	85.8 ^{d,f}	± 13.8	82.5 ^{d,f,§}	± 13.2				D x M = 0.60	
	HP	89.2	± 18.1	85.3 [§]	± 17.5	87.4	± 0.83	T x D = 0.002		
	HC	82.5 ^a	± 14.3	79.5 ^{a,§}	± 13.7	81.6	± 0.96	T x M = 0.070		
	AH	84.0	± 16.7	80.7 [§]	± 16.1	82.2	± 0.83	T x D x M = 0.94		
	MS	89.2 ^b	± 16.6	85.4 ^{b,§}	± 15.9	86.8	± 0.97			
	Time	86.2	± 16.9	82.7*	± 16.2					
Fat Mass (kg)	HP-AH	40.2	± 11.3	37.1 [§]	± 11.2				T = 0.001	
	HP-MS	42.2	± 11.6	39.0 [§]	± 11.2				D = 0.001	
	HC-AH	36.1 ^c	± 9.2	33.8 ^{c,§}	± 8.8				M = 0.001	
	HC-MS	38.7 ^{d,f}	± 8.7	36.0 ^{d,§}	± 8.3				D x M = 0.79	
	HP	41.1	± 11.4	38.0 [§]	± 11.2	39.6	± 0.53	T x D = 0.003		
	HC	37.1 ^a	± 9.1	34.7 ^{a,§}	± 8.7	36.2	± 0.61	T x M = 0.36		
	AH	38.2	± 10.5	35.5 [§]	± 10.2	36.8	± 0.53	T x D x M = 0.44		
	MS	40.8 ^b	± 10.7	37.9 ^{b,§}	± 10.2	39.0	± 0.62			
	Time	39.3	± 10.7	36.5*	± 10.3					
Lean Mass (kg)	HP-AH	45.3	± 7.9	44.7 [§]	± 7.6				T = 0.001	
	HP-MS	47.5 ^c	± 7.4	46.4 ^{c,§}	± 7.0				D = 0.001	
	HC-AH	42.5 ^c	± 6.3	41.9 ^{c,§}	± 6.1				M = 0.001	
	HC-MS	45.4 ^{d,f}	± 6.2	44.7 ^{d,f,§}	± 6.1				D x M = 0.40	
	HP	46.3	± 7.7	45.5 [§]	± 7.4	46.0	± 0.36	T x D = 0.18		
	HC	43.6 ^a	± 6.4	43.0 ^{a,§}	± 6.2	43.6	± 0.41	T x M = 0.07		
	AH	44.0	± 7.3	43.4 [§]	± 7.1	43.6	± 0.36	T x D x M = 0.27		
	MS	46.6 ^b	± 7.0	45.7 ^{b,§}	± 6.7	46.0	± 0.42			
	Time	45.1	± 7.3	44.4*	± 7.0					
Body Fat (%)	HP-AH	45.5	± 4.3	43.8 [§]	± 4.7				T = 0.001	
	HP-MS	45.5	± 4.5	44.1 [§]	± 4.7				D = 0.048	
	HC-AH	44.7	± 4.4	43.3 [§]	± 4.4				M = 0.75	
	HC-MS	44.8	± 3.8	43.4 [§]	± 4.0				D x M = 0.94	
	HP	45.5	± 4.4	43.9 [§]	± 4.7	44.7	± 0.23	T x D = 0.20		
	HC	44.7 ^a	± 4.2	43.4 [§]	± 4.3	44.0	± 0.26	T x M = 0.78		
	AH	45.1	± 4.4	43.6 [§]	± 4.6	44.3	± 0.22	T x D x M = 0.24		
	MS	45.2	± 4.3	43.8 [§]	± 4.5	44.4	± 0.26			
	Time	45.2	± 4.3	43.7*	± 4.5					
HP = higher protein, HC = higher carbohydrate, AH = apparently healthy, MS = metabolic syndrome risk factor, T = time effect, D = diet effect, M = metabolic syndrome risk factor effect, D x M = diet by metabolic syndrome risk factor effect, T x D = time by diet effect, T x M = time by metabolic syndrome risk factor effect, T x D x M = time by diet by metabolic syndrome risk factor effect. Values are means ± standard deviations (except group means are means ± standard error) from 198 participants in the HP-AH group, 173 in the HP-MS group, 179 in the HC-AH group, 113 in the HC-MS group, 371 in the HP total group, 292 in the HC total group, 377 in the AH group, 286 in the MS group, and 663 participants total. * Significantly different than baseline, p < 0.05 (univariate). † Significant diet effect, p < 0.05 (univariate). ‡ Significant metabolic syndrome effect, p < 0.05 (univariate). ^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than AH group, p < 0.05 (post hoc LSD). ^c Significantly different than HP-AH group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-MS group, p < 0.05 (post hoc LSD). ^e Significantly different than HC-AH group, p < 0.05 (post hoc LSD). ^f Significantly different than baseline, p < 0.05 (post hoc LSD).										

Changes in total scanned mass (HP -3.9 ± 3.5 , HC -3.0 ± 3.5 kg, $p=0.002$) and fat mass (HP -3.1 ± 2.7 , HC -2.4 ± 2.8 kg, $p=0.003$) were significantly greater in the HP group. When analyzed based on metabolic syndrome risk, those with MS tended to experience greater decreases in scanned mass (AH -3.9 ± 3.5 , MS -3.0 ± 3.5 kg, $p=0.07$) and lean mass (AH -0.55 ± 2.0 , MS -0.88 ± 2.0 kg, $p=0.07$). No significant group differences were observed for body fat percentage, and no significant time x diet x metabolic syndrome risk factor interactions were seen in any body composition variables measured via DEXA. Post hoc analysis of body composition data expressed in delta changes from baseline revealed a significantly greater decrease in scanned mass for HP-AH (-3.7 ± 3.5) than HC-AH (-2.8 ± 3.5 , $p=0.019$) and for HP-MS (-4.2 ± 3.5 kg) than HC-MS (-3.3 ± 3.5 , $p=0.036$). A significantly greater decrease in fat mass was observed for HP-AH (-3.1 ± 2.7 kg) than HC-AH (-2.3 ± 2.6 kg, $p=0.004$), as well as a significantly greater decrease in lean mass for HP-MS (-1.0 ± 2.1 kg) than HP-AH (-0.6 ± 1.9 kg, $p=0.025$). Figures 4.5 and 4.6 depict the delta change from baseline for each DEXA body composition variable for both the individual group comparisons and the group-combinations.

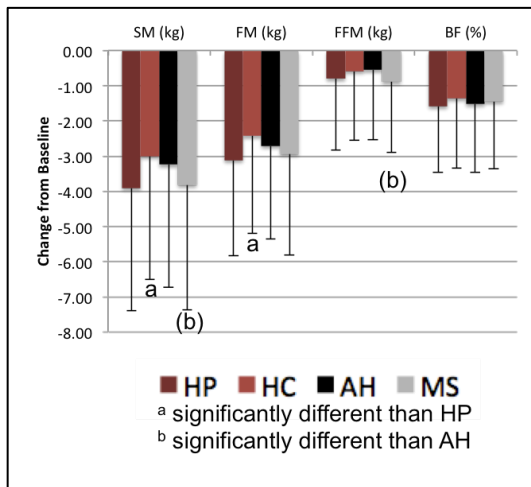


Figure 4.5: Delta Changes in DEXA Body Composition Variables, by Diet and Metabolic Syndrome Status

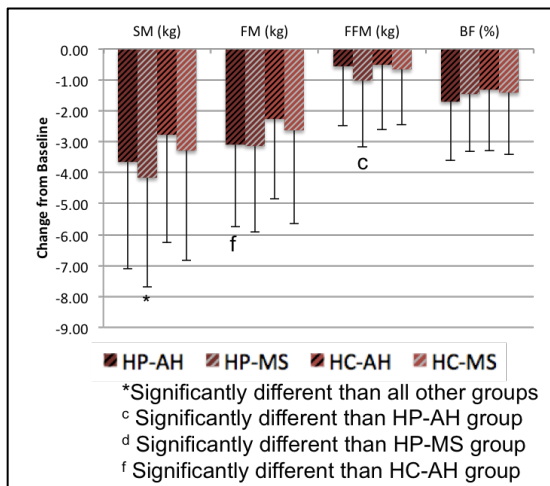


Figure 4.6: Delta Changes in DEXA Body Composition Variables, by Group-Combination

Anthropometric Measurements

Table 4.10 depicts the individual time x diet x metabolic syndrome risk factor ANOVAs for each of the anthropometric body composition variables. The variables included are weight (kg), body mass index (kg/m^2), and waist- and hip- circumference (cm), and are each discussed individually below.

In regards to weight, the three-way ANOVA revealed an overall time ($p<0.001$), time x diet ($p<0.001$) and time x metabolic syndrome risk factor ($p=0.054$) effect, with no significant time x diet x metabolic syndrome risk factor effect ($p=0.69$). Analysis of the ANOVA univariate test revealed a significant time effect (-3.8 ± 3.5 kg, $p<0.001$), with a significantly greater decrease in the HP group (HP -4.3 ± 3.6 , HC -3.2 ± 3.4 kg, $p=0.001$), and the MS group (AH -3.6 ± 3.4 , MS -4.2 ± 3.6 kg, $p=0.054$). Post hoc delta analysis of weight data expressed in delta changes from baseline revealed the HP-AH group lost significantly more weight (-4.1 ± 3.5 kg) than HC-AH (-3.0 ± 3.2 kg, $p=0.003$), and the HP-MS group lost significantly more weight (-4.5 ± 3.6 kg) than HC-MS (-3.6 ± 3.6 kg, $p=0.043$).

For body mass index, the three-way ANOVA revealed an overall time ($p<0.001$), time x diet ($p<0.001$) and time x metabolic syndrome risk factor ($p=0.046$) effect for BMI with no significant time x diet x metabolic syndrome risk factor effect ($p=0.74$). Analysis of the ANOVA univariate test revealed a significant time effect (-1.4 ± 1.3 kg/m^2 , $p<0.001$), with a significantly greater decrease in the HP group (HP -1.6 ± 1.3 , HC -1.2 ± 1.3 kg/m^2 , $p<0.001$), and the MS group (AH -1.3 ± 1.3 , MS -1.6 ± 1.3 kg/m^2 , $p=0.046$).

Table 4.10: Body Composition Before and After Ten Weeks of Exercise and Dietary Intervention Based on Metabolic Syndrome Status and Measured via Anthropometric Measurements

Variable	Group p	Baseline		10 Weeks			Group (SEM)			P-level
Weight (kg)	HP-AH	93.7	± 19.0	89.7 [§]	± 18.5				T = 0.001	
	HP-MS	98.1 ^c	± 18.9	93.6 ^{c,g}	± 18.0				D = 0.001	
	HC-AH	86.5 ^c	± 14.8	83.5 ^{c,g}	± 14.1				M = 0.001	
	HC-MS	92.3 ^{d,f}	± 14.4	88.7 ^{d,f,g}	± 13.9				D x M = 0.62	
	HP	95.8	± 19.1	91.5 [§]	± 18.3	93.8 [†]	± 0.9	T x D = 0.001		
	HC	88.8 ^a	± 14.9	85.5 ^{a,g}	± 14.2	87.8	± 1.0	T x M = 0.054		
	AH	90.3	± 17.5	86.7 [§]	± 16.8	88.3	± 0.9	T x D x M = 0.69		
	MS	95.8 ^b	± 17.5	91.7 ^{b,g}	± 16.6	93.2 [‡]	± 1.0			
Time	92.7	± 17.7	88.9 [*]	± 16.9						
Body Mass Index (kg/m ²)	HP-AH	35.0	± 6.6	33.5 [§]	± 6.4				T = 0.001	
	HP-MS	37.0 ^c	± 6.4	35.3 ^{c,g}	± 6.2				D = 0.001	
	HC-AH	32.5 ^c	± 5.3	31.4 ^{c,g}	± 5.0				M = 0.001	
	HC-MS	34.5 ^{d,f}	± 4.9	33.2 ^{d,f,g}	± 4.7				D x M = 0.98	
	HP	35.9	± 6.6	34.3 [§]	± 6.4	35.2 [†]	± 0.3	T x D = 0.001		
	HC	33.3 ^a	± 5.2	32.1 ^{a,g}	± 5.0	32.9	± 0.3	T x M = 0.046		
	AH	33.8	± 6.1	32.5 [§]	± 5.9	33.1	± 0.3	T x D x M = 0.74		
	MS	36.0 ^b	± 6.0	34.5 ^{b,g}	± 5.7	35.0 [‡]	± 0.4			
Time	34.8	± 6.2	33.3 [*]	± 5.9						
Waist Circumference (cm)	HP-AH	98.4	± 13.2	94.8 [§]	± 13.6				T = 0.001	
	HP-MS	104.7 ^c	± 13.0	100.2 ^{c,g}	± 12.1				D = 0.001	
	HC-AH	93.1 ^c	± 11.0	90.0 ^{c,g}	± 10.4				M = 0.001	
	HC-MS	100.0 ^{d,f}	± 11.4	96.7 ^{d,f,g}	± 11.3				D x M = 0.61	
	HP	101.3	± 13.5	97.3 [§]	± 13.2	99.5 [†]	± 0.6	T x D = 0.07		
	HC	95.8 ^a	± 11.7	92.6 ^{a,g}	± 11.2	95.0	± 0.7	T x M = 0.19		
	AH	95.9	± 12.5	92.5 [§]	± 12.4	94.1	± 0.6	T x D x M = 0.48		
	MS	102.9 ^b	± 12.6	98.8 ^{b,g}	± 11.9	100.4 [‡]	± 0.7			
Time	98.9	± 13.0	95.2 [*]	± 12.6						
Hip Circumference (cm)	HP-AH	121.8	± 13.5	118.4	± 13.5				T = 0.001	
	HP-MS	123.8	± 14.3	120.8	± 13.7				D = 0.001	
	HC-AH	116.7	± 11.8	114.6	± 11.0				M = 0.005	
	HC-MS	120.9	± 10.6	117.3	± 10.5				D x M = 0.53	
	HP	122.7	± 13.9	119.5 [§]	± 13.6	121.2 [†]	± 0.6	T x D = 0.40		
	HC	118.3 ^a	± 11.5	115.6 ^{a,g}	± 10.9	117.4	± 0.7	T x M = 0.19		
	AH	119.4	± 13.0	116.6 [§]	± 12.5	117.9	± 0.6	T x D x M = 0.022		
	MS	122.6 ^b	± 13.0	119.4 ^{b,g}	± 12.6	120.7 [‡]	± 0.7			
Time	120.8	± 13.1	117.8 [*]	± 12.6						
HP = higher protein, HC = higher carbohydrate, AH = apparently healthy, MS = metabolic syndrome risk factor, T = time effect, D = diet effect, M = metabolic syndrome risk factor effect, D x M = diet by metabolic syndrome risk factor effect, T x D = time by diet effect, T x M = time by metabolic syndrome risk factor effect, T x D x M = time by diet by metabolic syndrome risk factor effect. Values are means ± standard deviations (except group means are means ± standard error) from 198 participants in the HP-AH group, 173 in the HP-MS group, 179 in the HC-AH group, 113 in the HC-MS group, 371 in the HP total group, 292 in the HC total group, 377 in the AH group, 286 in the MS group, and 663 participants total. * Significantly different than baseline, p < 0.05 (univariate). † Significant diet effect, p < 0.05 (univariate). ‡ Significant metabolic syndrome effect, p < 0.05 (univariate). ^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than AH group, p < 0.05 (post hoc LSD). ^c Significantly different than HP-AH group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-MS group, p < 0.05 (post hoc LSD). ^e Significantly different than HC-AH group, p < 0.05 (post hoc LSD). ^f Significantly different than baseline, p < 0.05 (post hoc LSD).										

Post hoc delta analysis of body mass index data expressed in delta changes from baseline revealed a significantly greater decrease in BMI for HP-AH ($-1.5 \pm 1.3 \text{ kg/m}^2$) than HC-AH ($-1.1 \pm 1.2 \text{ kg/m}^2$, $p=0.003$), and a significantly greater decrease in BMI for HP-MS ($-1.7 \pm 1.3 \text{ kg/m}^2$) than HC-MS ($-1.4 \pm 1.3 \text{ kg/m}^2$, $p=0.036$).

In regards to waist circumference, the three-way ANOVA revealed an overall time effect ($p<0.001$) and a trend towards significance for time x diet ($p=0.07$). No significant time x metabolic syndrome risk factor ($p=0.19$) or time x diet x metabolic syndrome risk factor ($p=0.48$) interactions were observed. Overall, participants lost an average of $3.7 \pm 5.7 \text{ cm}$ in waist circumference throughout the ten-week protocol. When analyzed based on time x diet, those consuming the higher protein diet tended to experience a greater decrease in waist circumference (HP -4.0 ± 5.7 , HC $-3.2 \pm 5.7 \text{ cm}$, $p=0.07$). Post hoc analysis of waist circumference data expressed in delta changes from baseline revealed a significant difference from baseline for each of the group-combinations, but no other significant differences among groups.

For hip circumference, the three-way ANOVA revealed an overall time ($p<0.001$) and time x diet x metabolic syndrome risk factor ($p=0.022$) effect for hip circumference. Overall, participants lost an average of $3.0 \pm 5.1 \text{ cm}$ in hip circumference throughout the ten-week protocol, yet no significant time x diet ($p=0.40$) or time x metabolic syndrome risk factor ($p=0.19$) interactions were observed. Post hoc analysis of hip circumference data expressed in delta changes from baseline revealed that HC-AH lost significantly less centimeters (-2.1 ± 5.0) than both HP-AH (-3.4 ± 5.2 , $p=0.016$) and HC-MS (-3.6 ± 4.8 , $p=0.017$).

Figures 4.7 and 4.8 depict the delta change from baseline for each anthropometric body composition variable for both the individual group comparisons and the group-combinations. Based on these findings, hypothesis H₃, which indicated there would be statistically significant differences in the changes of body composition as a result of diet intervention and/or the presence of metabolic syndrome risk status was accepted.

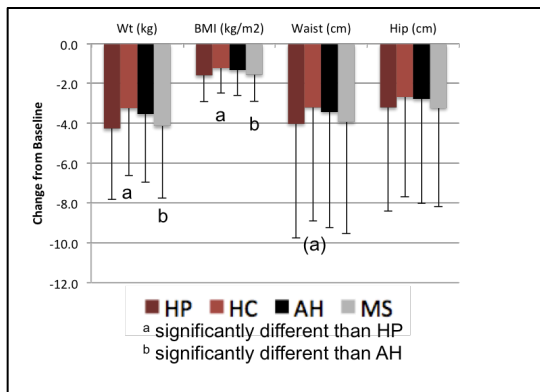


Figure 4.7: Delta Changes in Anthropometric Body Composition Variables, by Diet and Metabolic Syndrome Status

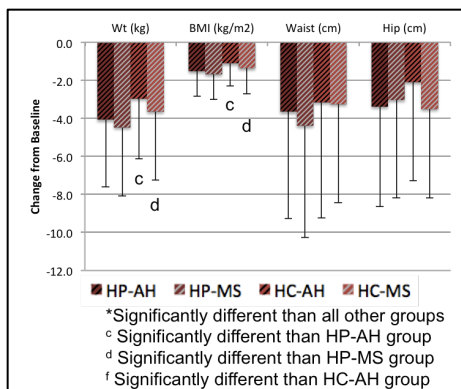


Figure 4.8: Delta Changes in Anthropometric Body Composition Variables, by Group-Combination

Resting Energy Expenditure

Table 4.11 depicts the time x diet x metabolic syndrome risk factor ANOVA for resting energy expenditure (REE), which was measured on a subset of 633 participants. The three-way ANOVA revealed an overall time ($p < 0.001$) effect for resting energy expenditure. No significant time x diet ($p = 0.30$), time x metabolic syndrome risk factor ($p = 0.88$), or time x diet x metabolic syndrome risk factor ($p = 0.98$) interactions were observed. Overall, participants lost an average of 55.6 ± 212 kcal/day in resting energy expenditure throughout the ten-week protocol, yet no significant differences were observed over-time based on diet protocol or health status. Post hoc analysis of resting energy expenditure data on delta calculations between testing sessions shows a significant change from baseline for all four group-combinations, but no significant differences between the groups. Therefore, hypothesis H_4 is accepted, which indicated there would not be statistically significant differences in resting energy expenditure parameters as a result of diet intervention and/or the presence of metabolic syndrome risk status.

Table 4.11: Resting Energy Expenditure After Ten Weeks of Exercise and Dietary Intervention Based on Metabolic Syndrome Status

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Resting Energy Expenditure (kcal/day)	HP-AH	1639.8	± 272.6	1577.2 ^g	± 269.9				T = 0.001
	HP-MS	1761.4 ^c	± 286.1	1696.7 ^{c,g}	± 304.3				D = 0.001
	HC-AH	1562.5 ^c	± 239.4	1518.5 ^{c,g}	± 246.4				M = 0.001
	HC-MS	1661.8 ^{d,f}	± 250.4	1614.7 ^{d,f,g}	± 242.2				D x M = 0.57
	HP	1697.4	± 285.2	1633.8 ^g	± 292.5	1668.8 [†]	± 13.0		T x D = 0.30
	HC	1600.7 ^a	± 248.0	1555.5 ^{a,g}	± 248.8	1589.4	± 15.1		T x M = 0.88
	AH	1603.1	± 259.9	1549.4 ^b	± 260.3	1574.5	± 13.0		T x D x M = 0.98
	MS	1723.0 ^b	± 276.7	1665.1 ^{b,g}	± 284.3	1683.7 [‡]	± 15.2		
	Time	1655.2	± 273.7	1599.7 [*]	± 276.8				

HP = higher protein, HC = higher carbohydrate, AH = apparently healthy, MS = metabolic syndrome risk factor, T = time effect, D = diet effect, M = metabolic syndrome risk factor effect, D x M = diet by metabolic syndrome risk factor effect, T x D = time by diet effect, T x M = time by metabolic syndrome risk factor effect, T x D x M = time by diet by metabolic syndrome risk factor effect.
Resting energy expenditure values are means ± standard deviations (except group means are means ± standard error) from 181 participants in the HP-AH group, 176 in the HP-MS group, 163 in the HC-AH group, 113 in the HC-MS group, 357 in the HP total group, 276 in the HC total group, 344 in the AH group, 289 in the MS group, and 633 participants total.
^{*} Significantly different than baseline, $p < 0.05$ (univariate). [†] Significant diet effect, $p < 0.05$ (univariate). [‡] Significant metabolic syndrome effect, $p < 0.05$ (univariate). ^a Significantly different than HP group, $p < 0.05$ (post hoc LSD). ^b Significantly different than AH group, $p < 0.05$ (post hoc LSD). ^c Significantly different than HP-AH group, $p < 0.05$ (post hoc LSD). ^d Significantly different than HP-MS group, $p < 0.05$ (post hoc LSD). ^f Significantly different than HC-AH group, $p < 0.05$ (post hoc LSD). ^g Significantly different than baseline, $p < 0.05$ (post hoc LSD).

Hemodynamic Variables

Table 4.12 depicts the time x diet x metabolic syndrome risk factor MANOVA for resting hemodynamic measurements including resting heart rate, systolic, and diastolic blood pressure. The three-way MANOVA revealed an overall time (Wilks' Lambda $p < 0.001$) and time x metabolic syndrome risk factor (Wilks' Lambda $p < 0.001$) effect for resting hemodynamic parameters, but no significant time x diet (Wilks' Lambda $p = 0.73$) or time x diet x metabolic syndrome risk factor (Wilks' Lambda $p = 0.83$) interactions.

Overall, resting heart rate decreased by 3.0 ± 10.3 bpm (time effect $p < 0.001$) throughout the ten-week study, however no significant changes were observed over time based on a specific dietary protocol or health status. Additionally, post hoc delta analysis shows a significant change from baseline for each of the four group-combinations, but no significant differences between the groups.

Table 4.12: Resting Hemodynamic Measurements After Ten Weeks of Exercise and Dietary Intervention Based on Metabolic Syndrome Status

Variable	Group	Baseline			10 Weeks			Group (SEM)			P-level
Resting Heart Rate (bpm)	HP-AH	71.0	±	9.8	68.6 ^g	±	10.0				T = 0.001
	HP-MS	73.2 ^c	±	10.8	69.3 ^g	±	9.3				D = 0.44
	HC-AH	70.0	±	9.8	67.5 ^g	±	10.1				M = 0.005
	HC-MS	73.1 ^f	±	11.3	69.4 ^g	±	9.7				D x M = 0.46
	HP	72.0	±	10.3	68.9 ^g	±	9.7	70.5	±	0.5	T x D = 0.91
	HC	71.2	±	10.5	68.2 ^g	±	9.9	70.0	±	0.5	T x M = 0.10
	AH	70.5	±	9.8	68.1 ^g	±	10.0	69.3	±	0.4	T x D x M = 0.91
	MS	73.1 ^b	±	11.0	69.3 ^g	±	9.4	71.2 [‡]	±	0.5	
	Time	71.6	±	10.4	68.6 [*]	±	9.8				
Resting Systolic Blood Pressure (mmHg)	HP-AH	121.3	±	12.6	121.4	±	13.3				T = 0.001
	HP-MS	130.4 ^c	±	15.5	124.8 ^{c,g}	±	14.0				D = 0.33
	HC-AH	120.5	±	11.9	119.4	±	12.9				M = 0.001
	HC-MS	130.3 ^f	±	16.0	124.0 ^{f,g}	±	15.5				D x M = 0.60
	HP	125.6	±	14.7	123.0 ^g	±	13.7	124.5	±	0.6	T x D = 0.43
	HC	124.3	±	14.4	121.2 ^g	±	14.1	123.6	±	0.7	T x M = 0.001
	AH	120.9	±	12.2	120.5	±	13.2	120.7	±	0.6	T x D x M = 0.87
	MS	130.4 ^b	±	15.7	124.4 ^{b,g}	±	14.6	127.4 [‡]	±	0.7	
	Time	125.0	±	14.6	122.2 [*]	±	13.9				
Resting Diastolic Blood Pressure (mmHg)	HP-AH	78.6	±	8.1	79.0	±	8.9				T = 0.001
	HP-MS	84.3 ^c	±	9.6	80.2 ^g	±	8.7				D = 0.001
	HC-AH	77.3	±	8.6	76.1 ^c	±	8.9				M = 0.001
	HC-MS	82.6 ^f	±	9.6	78.5 ^{f,g}	±	9.5				D x M = 0.72
	HP	81.3	±	9.3	79.6 ^g	±	8.8	80.5 [†]	±	0.4	T x D = 0.28
	HC	79.3 ^a	±	9.4	77.0 ^{a,g}	±	9.2	78.6	±	0.5	T x M = 0.001
	AH	78.0	±	8.4	77.6	±	9.0	77.7	±	0.4	T x D x M = 0.38
	MS	83.6 ^b	±	9.6	79.5 ^{b,g}	±	9.0	81.4 [‡]	±	0.5	
	Time	80.4	±	9.4	78.4 [*]	±	9.1				

HP = higher protein, HC = higher carbohydrate, AH = apparently healthy, MS = metabolic syndrome risk factor, T = time effect, D = diet effect, M = metabolic syndrome risk factor effect, D x M = diet by metabolic syndrome risk factor effect, T x D = time by diet effect, T x M = time by metabolic syndrome risk factor effect, T x D x M = time by diet by metabolic syndrome risk factor effect.
Resting hemodynamic values are means ± standard deviations (except group means are means ± standard error) from 198 participants in the HP-AH group, 173 in the HP-MS group, 179 in the HC-AH group, 113 in the HC-MS group, 371 in the HP total group, 292 in the HC total group, 377 in the AH group, 286 in the MS group, and 663 participants total.
^{*} Significantly different than baseline, p < 0.05 (univariate). [†] Significant diet effect, p < 0.05 (univariate). [‡] Significant metabolic syndrome effect, p < 0.05 (univariate). ^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than AH group, p < 0.05 (post hoc LSD). ^c Significantly different than HP-AH group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-MS group, p < 0.05 (post hoc LSD). ^e Significantly different than HC-AH group, p < 0.05 (post hoc LSD). ^f Significantly different than baseline, p < 0.05 (post hoc LSD).

In regards to blood pressure, over the course of the study, participants observed an average decrease in systolic blood pressure by 2.8±14.8 mmHg (time effect, p<0.001) and diastolic blood pressure by 2.0±9.8 mmHg (time effect, p<0.001). While no significant differences were seen overtime based on diet, the MS group experienced a

significantly greater decrease in systolic blood pressure (AH -0.5 ± 13.3 , MS -5.9 ± 16.0 mmHg, $p < 0.001$) and diastolic blood pressure (AH -0.4 ± 8.9 , MS -4.1 ± 10.5 mmHg, $p < 0.001$). For both systolic and diastolic blood pressure, post hoc analysis on delta changes from baseline revealed that the HP-MS and HC-MS groups both demonstrated significant decreases from baseline (each with $p < 0.001$).

The HP-MS group experienced a significantly greater decrease in blood pressure than the HP-AH group (SBP: HP-AH $+0.5 \pm 13.2$, HP-MS -5.6 ± 15.6 mmHg, $p < 0.001$), (DBP: HP-AH $+0.4 \pm 8.8$, HP-MS -4.0 ± 10.4 mmHg, $p < 0.001$). The HC-MS group experienced a significantly greater decrease in blood pressure than the HC-AH group (SBP: HC-AH -1.1 ± 13.4 , HC-MS -6.3 ± 16.6 mmHg, $p = 0.003$), (DBP: HC-AH -1.1 ± 9.0 , HC-MS -4.2 ± 10.7 mmHg, $p = 0.009$).

Figures 4.9 and 4.10 depict the delta changes for the hemodynamic parameters over the ten week study. Based on the time x metabolic syndrome risk factor effects for both SBP and DBP, hypothesis H_5 , which indicated there would not be statistically significant differences in resting hemodynamic parameters as a result of diet intervention and/or the presence of metabolic syndrome risk status, is rejected.

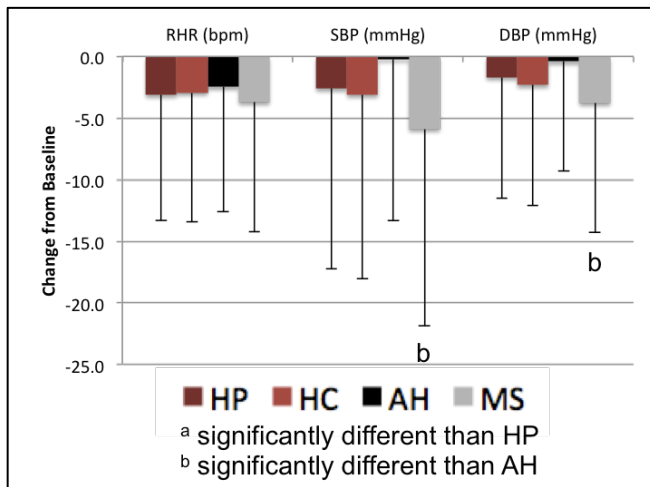


Figure 4.9: Delta Changes in Hemodynamic Variables, by Diet and Metabolic Syndrome Status

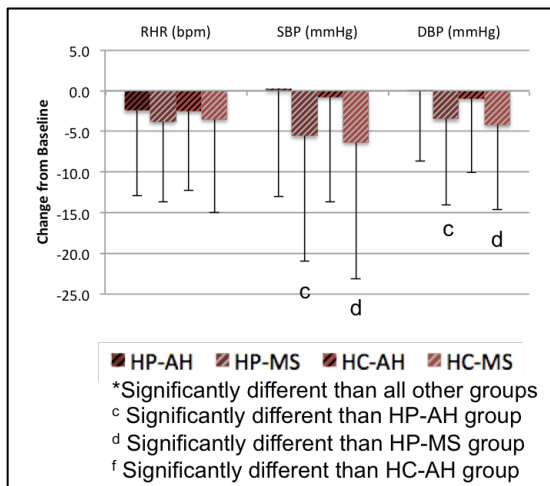


Figure 4.10: Delta Changes in Hemodynamic Variables, by Group-Combination

Blood Lipids

Table 4.13 depicts the time x diet x metabolic syndrome risk factor MANOVA for blood lipid values. The variables reported that will each be discussed individually include total cholesterol (mmol/L), LDL cholesterol (mmol/L), HDL cholesterol (mmol/L), Triglycerides (mmol/L), and TC/HDL ratio. The three-way MANOVA revealed a significant overall time (Wilks' Lambda $p<0.001$) and time x metabolic syndrome risk factor (Wilks' Lambda $p<0.001$) effect as well as a trend for a time x diet x metabolic syndrome risk factor (Wilks' Lambda $p=0.09$) effect for blood lipids. No significant time x diet (Wilks' Lambda $p=0.50$) interaction was observed.

In regards to total cholesterol, participants lost an average of 0.2 ± 0.8 mmol/L (time effect, $p<0.001$) throughout the study protocol, however no significant diet or health status effects were reported over time. Significant changes from baseline were observed through post hoc analysis on blood lipid data expressed in delta changes from baseline for the HP-AH ($p=0.002$), HP-MS ($p<0.001$), and HC-AH ($p=0.005$) groups. No significant differences were observed between the group-combinations in the post-hoc analysis.

For LDL cholesterol, participants lost an average of 0.14 ± 0.6 mmol/L (time effect, $p<0.001$) throughout the study protocol, however no significant diet or health status effects were reported over time. Significant changes from baseline were observed through post hoc analysis on blood lipid data expressed in delta changes from baseline for the HP-AH ($p=0.003$), HP-MS ($p<0.001$), and HC-AH ($p=0.001$) groups.

Table 4.13: Fasting Blood Lipid Values After Ten Weeks of Exercise and Dietary Intervention Based on Metabolic Syndrome Status

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Total Cholesterol (mmol/L)	HP-AH	5.1	± 0.9	5.0 ^g	± 0.9				T = 0.001
	HP-MS	5.3	± 1.0	5.0 ^g	± 1.0				D = 0.38
	HC-AH	5.1	± 1.0	4.9 ^g	± 0.9				M = 0.001
	HC-MS	5.4 ^f	± 1.1	5.3 ^{d,f}	± 1.0				D x M = 0.11
	HP	5.2	± 1.0	5.0 ^g	± 1.0	5.1	± 0.05		T x D = 0.40
	HC	5.2	± 1.0	5.0 ^g	± 1.0	5.2	± 0.05		T x M = 0.76
	AH	5.1	± 0.9	4.9 ^g	± 0.9	5.0	± 0.05		T x D x M = 0.48
	MS	5.3 ^b	± 1.1	5.1 ^{b,g}	± 1.0	5.2 [‡]	± 0.05		
	Time	5.2	± 1.0	5.0*	± 1.0				
LDL (mmol/L)	HP-AH	3.0	± 0.7	2.9 ^g	± 0.7				T = 0.001
	HP-MS	3.2 ^c	± 0.8	3.0 ^g	± 0.8				D = 0.19
	HC-AH	3.0	± 0.8	2.8 ^g	± 0.8				M = 0.001
	HC-MS	3.3 ^f	± 0.9	3.3 ^{d,f}	± 0.8				D x M = 0.047
	HP	3.1	± 0.8	3.0 ^g	± 0.8	3.0	± 0.04		T x D = 0.53
	HC	3.1	± 0.8	3.0 ^g	± 0.8	3.1	± 0.04		T x M = 0.64
	AH	3.0	± 0.8	2.9 ^g	± 0.7	2.9	± 0.04		T x D x M = 0.26
	MS	3.3 ^b	± 0.8	3.1 ^{b,g}	± 0.8	3.2 [‡]	± 0.04		
	Time	3.1	± 0.8	3.0*	± 0.8				
HDL (mmol/L)	HP-AH	1.5	± 0.3	1.4 ^g	± 0.3				T = 0.001
	HP-MS	1.2 ^c	± 0.3	1.2 ^{c,g}	± 0.3				D = 0.70
	HC-AH	1.5	± 0.3	1.4 ^g	± 0.3				M = 0.001
	HC-MS	1.2 ^f	± 0.3	1.2 ^{f,g}	± 0.3				D x M = 0.63
	HP	1.4	± 0.3	1.3 ^g	± 0.3	1.3	± 0.02		T x D = 0.71
	HC	1.4	± 0.3	1.3 ^g	± 0.3	1.3	± 0.02		T x M = 0.016
	AH	1.5	± 0.3	1.4 ^g	± 0.3	1.5	± 0.01		T x D x M = 0.40
	MS	1.2 ^b	± 0.3	1.2 ^{b,g}	± 0.3	1.2 [‡]	± 0.02		
	Time	1.4	± 0.3	1.3*	± 0.3				
Triglycerides (mmol/L)	HP-AH	1.2	± 0.5	1.2	± 0.6				T = 0.001
	HP-MS	2.0 ^c	± 1.0	1.8 ^{c,g}	± 0.9				D = 0.83
	HC-AH	1.2	± 0.5	1.3	± 0.6				M = 0.001
	HC-MS	2.0 ^f	± 0.8	1.8 ^{f,g}	± 0.8				D x M = 0.72
	HP	1.6	± 0.8	1.5 ^g	± 0.8	1.6	± 0.03		T x D = 0.13
	HC	1.5	± 0.7	1.5 ^g	± 0.7	1.6	± 0.04		T x M = 0.001
	AH	1.2	± 0.5	1.2	± 0.6	1.2	± 0.03		T x D x M = 0.001
	MS	2.0 ^b	± 0.9	1.8 ^{b,g}	± 0.9	1.9 [‡]	± 0.04		
	Time	1.6	± 0.8	1.5*	± 0.8				
TC/HDL ratio	HP-AH	3.5	± 0.7	3.6 ^g	± 0.7				T = 0.026
	HP-MS	4.4 ^c	± 0.9	4.3 ^c	± 1.0				D = 0.22
	HC-AH	3.5	± 0.8	3.6	± 0.8				M = 0.001
	HC-MS	4.4 ^f	± 1.0	4.6 ^{d,f,g}	± 1.1				D x M = 0.24
	HP	3.9	± 0.9	3.9	± 0.9	4.0	± 0.04		T x D = 0.42
	HC	3.9	± 1.0	4.0 ^g	± 1.1	4.0	± 0.05		T x M = 0.41
	AH	3.5	± 0.8	3.6 ^g	± 0.8	3.6	± 0.04		T x D x M = 0.027
	MS	4.4 ^b	± 0.9	4.4 ^b	± 1.0	4.4 [‡]	± 0.05		
	Time	3.9	± 0.9	4.0*	± 1.0				

HP = higher protein, HC = higher carbohydrate, AH = apparently healthy, MS = metabolic syndrome risk factor, T = time effect, D = diet effect, M = metabolic syndrome risk factor effect, D x M = diet by metabolic syndrome risk factor effect, T x D = time by diet effect, T x M = time by metabolic syndrome risk factor effect, T x D x M = time by diet by metabolic syndrome risk factor effect.

Values are means ± standard deviations (except group means are means ± standard error) from 198 participants in the HP-AH group, 173 in the HP-MS group, 179 in the HC-AH group, 113 in the HC-MS group, 371 in the HP total group, 292 in the HC total group, 377 in the AH group, 286 in the MS group, and 663 participants total.

* Significantly different than baseline, $p < 0.05$ (univariate). ‡ Significant metabolic syndrome effect, $p < 0.05$ (univariate).

^a Significantly different than HP group, $p < 0.05$ (post hoc LSD). ^b Significantly different than AH group, $p < 0.05$ (post hoc LSD).

^c Significantly different than HP-AH group, $p < 0.05$ (post hoc LSD). ^d Significantly different than HP-MS group, $p < 0.05$ (post hoc LSD). ^e

Significantly different than HC-AH group, $p < 0.05$ (post hoc LSD). ^g Significantly different than baseline, $p < 0.05$ (post hoc LSD).

No significant differences were observed between the group-combinations in the post-hoc analysis.

In regards to HDL cholesterol, participants lost an average of 0.07 ± 0.2 mmol/L (time effect, $p < 0.001$) throughout the study protocol. While no effect was observed regarding dietary assignment over time, the AH group experienced a significantly greater decrease in HDL (AH -0.09 ± 0.3 , MS -0.04 ± 0.2 mmol/L, $p = 0.016$). Post hoc analysis using delta change shows a significant decrease from baseline for all four of the group-combinations, and also that the HP-AH group lost significantly more HDL (-0.1 ± 0.2 mmol/L) compared to HP-MS (-0.04 ± 0.2 mmol/L, $p = 0.013$).

For triglycerides, participants lost an average of 0.10 ± 0.6 mmol/L (time effect, $p < 0.001$) throughout the study protocol. No time x diet interaction was observed, however the MS group experienced a significantly greater decrease in triglycerides (AH -0.00 ± 0.47 , MS -0.22 ± 0.73 mmol/L, delta $p < 0.001$). Post hoc analyses based on delta change from baseline demonstrates a significant difference between HC-AH and all other group combinations, in that HC-AH was the only group that *increased* ($+0.06 \pm 0.5$ mmol/L) in triglyceride value (delta $p = 0.052$), however this value is not a significant increase from baseline. Figures 4.11 and 4.12 depict the delta changes for the blood lipid parameters over the ten-week period.

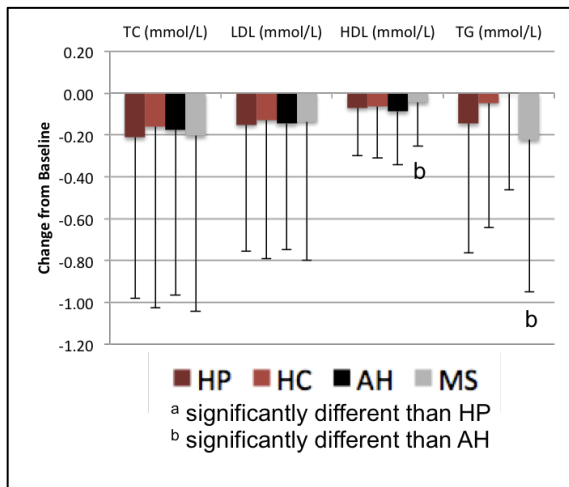


Figure 4.11: Delta Changes in Blood Lipid Variables, by Diet and Metabolic Syndrome Status

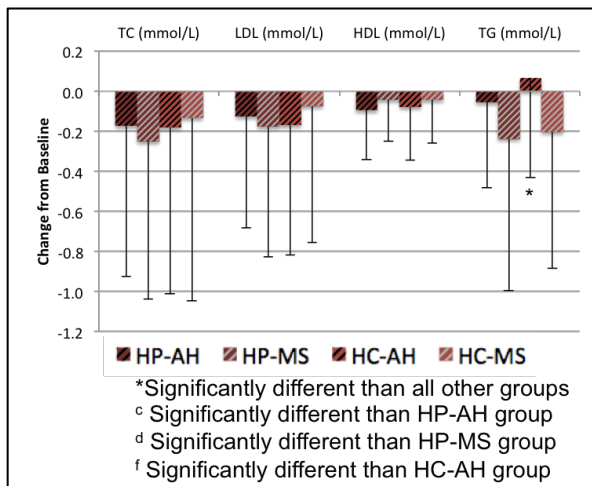


Figure 4.12: Delta Changes in Blood Lipid Variables, by Group-Combination

In regards to TC/HDL ratio, participants gained an average of 0.05 ± 0.7 in value (time effect, $p=0.026$), however no significant diet or health status effects were reported over time. Time x diet x metabolic syndrome risk factor analysis revealed a significant

three-way interaction ($p=0.027$). Analysis using delta changes from baseline revealed the HP-MS group to be significantly different from all other group combinations, in that all of the other groups *increased* in TC/HDL ratio over time, and HP-MS is the only group showing a *decrease* (-0.05 ± 0.73 , $p=0.035$), however this decrease was not a significant change in value from baseline.

Upon observing the decrease in HDL cholesterol for all groups, and most significantly for the apparently healthy participants, a three-way ANOVA of TG/HDL ratio (using mmol/L values) was added in, as is depicted in Table 4.14. A time x metabolic syndrome risk factor ($p<0.001$) effect, but only a trend in time ($p=0.07$), and no time x diet (0.38), or time x diet x metabolic syndrome risk factor ($p=0.57$) interactions were observed. Participants in the MS group had significantly greater TG/HDL ratios both at baseline and at ten-weeks, however the MS group also experienced a significant decrease compared to an increase in the AH group (AH $+0.05\pm0.42$, MS -0.14 ± 0.78 , $p<0.001$). Post hoc analysis using the delta change from baseline reveals that three of the groups-combinations experienced a significant change from baseline (HP-MS $p=0.001$; HC-AH $p=0.052$; and HC-MS $p=0.019$), with HP-MS (-0.14 ± 0.8) experiencing a significantly greater decrease than HP-AH ($+0.02\pm0.4$, $p=0.008$) and HC-MS (-0.13 ± 0.7) experiencing a significantly greater decrease than HC-AH ($+0.09\pm0.4$, $p=0.002$).

Table 4.14: Calculated TG/HDL Ratio After Ten Weeks of Exercise and Dietary Intervention Based on Metabolic Syndrome Status

Variable	Group	Baseline			10 Weeks			Group (SEM)	P-level
TG/HDL (mmol)	HP-AH	0.9	±	0.4	0.9	±	0.5		T = 0.07
	HP-MS	1.7 ^c	±	0.9	1.6 ^{c,g}	±	0.9		D = 0.88
	HC-AH	0.9	±	0.4	1.0 ^g	±	0.5		M = 0.001
	HC-MS	1.7 ^f	±	0.8	1.6 ^{f,g}	±	0.7		D x M = 0.51
	HP	1.3	±	0.8	1.2 ^g	±	0.8	1.3 ± 0.03	T x D = 0.39
	HC	1.2	±	0.7	1.2	±	0.7	1.3 ± 0.04	T x M = 0.001
	AH	0.9	±	0.4	0.9 ^g	±	0.5	0.9 ± 0.03	T x D x M = 0.57
	MS	1.7 ^b	±	0.9	1.6 ^{b,g}	±	0.9	1.6 [‡] ± 0.04	
	Time	1.2	±	0.8	1.2	±	0.8		

HP = higher protein, HC = higher carbohydrate, AH = apparently healthy, MS = metabolic syndrome risk factor, T = time effect, D = diet effect, M = metabolic syndrome risk factor effect, D x M = diet by metabolic syndrome risk factor effect, T x D = time by diet effect, T x M = time by metabolic syndrome risk factor effect, T x D x M = time by diet by metabolic syndrome risk factor effect.
Resting energy expenditure values are means ± standard deviations (except group means are means ± standard error) from 198 participants in the HP-AH group, 173 in the HP-MS group, 179 in the HC-AH group, 113 in the HC-MS group, 371 in the HP total group, 292 in the HC total group, 377 in the AH group, 286 in the MS group, and 663 participants total.
* Significantly different than baseline, p < 0.05 (univariate). † Significant diet effect, p < 0.05 (univariate). ‡ Significant metabolic syndrome effect, p < 0.05 (univariate). ^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than AH group, p < 0.05 (post hoc LSD).
^c Significantly different than HP-AH group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-MS group, p < 0.05 (post hoc LSD).
^f Significantly different than HC-AH group, p < 0.05 (post hoc LSD). ^g Significantly different than baseline, p < 0.05 (post hoc LSD).

Figures 4.13 and 4.14 depict the delta changes in the TC/HDL and TG/HDL ratios over the ten-week period. Based on the findings from Table 4.13, hypothesis H₆ is accepted. This hypothesis indicated there would be statistically significant differences in the changes in blood lipids as a result of diet intervention and/or the presence of metabolic syndrome risk status.

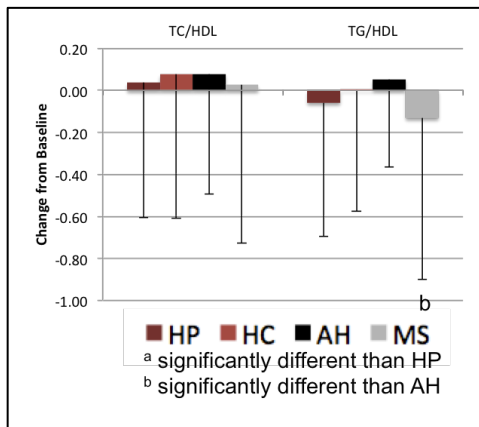


Figure 4.13: Delta Changes in Blood Lipid Ratios, by Diet and Metabolic Syndrome Status

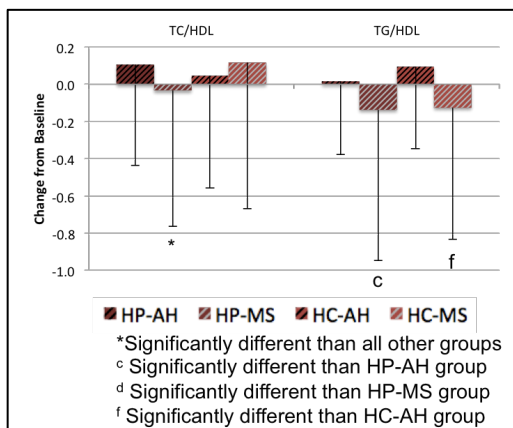


Figure 4.14: Delta Changes in Blood Lipid Variables, by Group-Combination

Glucose Homeostasis

Table 4.15 depicts the individual time x diet x metabolic syndrome risk factor ANOVAs for markers of glucose homeostasis including glucose, insulin, and calculated HOMA (Homeostatic Model Assessment). Each of these variables is discussed individually below.

Table 4.15: Fasting Glucose and Insulin Values After Ten Weeks of Exercise and Dietary Intervention Based on Metabolic Syndrome Status

Variable	Group	Baseline			10 Weeks			Group (SEM)			P-level
Glucose (mmol/L)	HP-AH	5.3	±	0.8	5.3	±	0.7				T = 0.003
	HP-MS	6.2 ^c	±	1.5	6.0 ^{c,g}	±	1.5				D = 0.64
	HC-AH	5.3	±	0.8	5.4	±	0.7				M = 0.001
	HC-MS	6.1 ^f	±	1.7	5.8 ^{f,g}	±	1.3				D x M = 0.24
	HP	5.7	±	1.2	5.6 ^g	±	1.2	5.7	±	0.05	T x D = 0.64
	HC	5.6	±	1.3	5.5	±	1.0	5.6	±	0.06	T x M = 0.001
	AH	5.3	±	0.8	5.3	±	0.7	5.3	±	0.05	T x D x M = 0.48
	MS	6.1 ^b	±	1.6	5.9 ^{b,g}	±	1.4	6.0‡	±	0.06	
	Time	5.7	±	1.3	5.6*	±	1.1				
Insulin (uIU/mL)	HP-AH	3.3	±	8.5	3.8	±	9.4				T = 0.86
	HP-MS	4.9	±	9.1	5.5	±	10.9				D =0.001
	HC-AH	5.8	±	6.6	7.8 ^c	±	11.5				M = 0.025
	HC-MS	12.1 ^{d,f}	±	18.4	9.5	±	12.8				D x M = 0.36
	HP	4.0	±	8.8	4.5	±	10.1	4.4†	±	0.80	T x D = 0.50
	HC	8.3 ^a	±	12.8	8.4 ^a	±	12.0	8.8	±	0.97	T x M = 0.07
	AH	4.4	±	7.8	5.5	±	10.5	5.2	±	0.80	T x D x M = 0.06
	MS	7.7 ^b	±	13.8	7.0	±	11.8	8.0‡	±	0.97	
	Time	5.7	±	10.8	6.1	±	11.1				
Calculated HOMA	HP-AH	0.8	±	2.2	0.9	±	2.3				T = 0.85
	HP-MS	1.3	±	2.5	1.6	±	3.5				D = 0.001
	HC-AH	1.4	±	1.5	2.0 ^c	±	3.5				M = 0.005
	HC-MS	3.8 ^{d,f}	±	6.9	2.6 ^g	±	3.5				D x M = 0.23
	HP	1.0	±	2.3	1.2	±	2.9	1.2†	±	0.23	T x D = 0.33
	HC	2.3 ^a	±	4.6	2.2 ^a	±	3.5	2.4	±	0.28	T x M = 0.06
	AH	1.1	±	2.0	1.4	±	2.9	1.3	±	0.23	T x D x M = 0.027
	MS	2.3 ^b	±	4.8	2.0	±	3.5	2.3‡	±	0.28	
	Time	1.6	±	3.5	1.6	±	3.2				
HP = higher protein, HC = higher carbohydrate, AH = apparently healthy, MS = metabolic syndrome risk factor T = time effect, D = diet effect, M = metabolic syndrome risk factor effect, D x M = diet by metabolic syndrome risk factor effect, T x D = time by diet effect, T x M = time by metabolic syndrome risk factor effect, T x D x M = time by diet by metabolic syndrome risk factor effect. Glucose values are means ± standard deviations (except group means are means ± standard error) from 198 participants in the HP-AH group, 173 in the HP-MS group, 179 in the HC-AH group, 113 in the HC-MS group, 371 in the HP total group, 292 in the HC total group, 377 in the AH group, 286 in the MS group, and 663 participants total. Insulin and HOMA values are means ± standard deviations (except group means are means ± standard error) from 80 participants in the HP-AH group, 69 in the HP-MS group, 58 in the HC-AH group, 45 in the HC-MS group, 149 in the HP total group, 103 in the HC total group, 138 in the AH group, 114 in the MS group, and 252 participants total. * Significantly different than baseline, p < 0.05 (univariate). † Significant diet effect, p < 0.05 (univariate). ‡ Significant metabolic syndrome effect, p < 0.05 (univariate). ^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than AH group, p < 0.05 (post hoc LSD). ^c Significantly different than HP-AH group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-MS group, p < 0.05 (post hoc LSD). ^e Significantly different than HC-AH group, p < 0.05 (post hoc LSD). ^f Significantly different than baseline, p < 0.05 (post hoc LSD).											

For glucose, the three-way ANOVA revealed an overall time effect (p=0.003) and time x metabolic syndrome risk factor effect (p=0.001). No significant time x diet (p=0.64) or time x diet x metabolic syndrome risk factor (p=0.48) interactions were observed. Overall, participants lost an average of 0.10±1.0 mmol/L (time effect, p=0.003) in fasting glucose throughout the study protocol. No significant effects were

observed overtime based on diet. Based on analysis using delta change calculations from baseline, the MS group experienced a significant decrease in glucose whereas the AH group did not experience a significant change from baseline (AH $+0.01 \pm 0.73$ mmol/L, MS -0.24 ± 1.19 mmol/L, $p=0.001$). Post hoc analysis on the delta change from baseline revealed that HP-MS and HC-MS were the only groups to significantly change from baseline ($p=0.002$ and $p=0.006$ respectively), and that HP-MS experienced significantly greater decreases than HP-AH (HP-AH -0.03 ± 0.8 , HP-MS -0.23 ± 1.2 , $p=0.051$) and HC-MS experienced a significantly greater decrease than HC-AH (HC-AH $+0.06 \pm 0.7$, HC-MS -0.24 ± 1.19 , $p=0.009$).

Insulin was analyzed on a subset of 252 participants from which serum was measured within the study. The three-way ANOVA revealed a trend for both time x metabolic syndrome risk factor ($p=0.07$) and time x diet x metabolic syndrome risk factor ($p=0.06$) effect for insulin. No overall time effect ($p=0.86$) or a time x diet ($p=0.50$) interactions were observed. Analysis on data calculated from delta change from baseline revealed a tendency for an *increase* in insulin for the AH group and a decrease in the MS group over time (AH $+1.11 \pm 6.5$ uIU/mL, MS -0.65 ± 12.2 uIU/mL, $p=0.07$) however these values were not significantly different from baseline, nor were the ten-week values for each group significantly different from one another. A trend was also observed in the time x diet x metabolic syndrome risk factor three-way interaction ($p=0.06$). Post hoc analysis on insulin data expressed in delta changes from baseline revealed a significant *increase* in insulin for the HC-AH group ($+1.9 \pm 9.4$ uIU/mL) and a

decrease in the HC-MS group (-2.5 ± 18.6 uIU/mL, $p=0.017$) though the decrease was not significantly different from baseline.

Homeostatic Model Assessment was calculated and analyzed on the subset of 252 participants for whom insulin measurements were obtained. The three-way ANOVA revealed an overall time x diet x metabolic syndrome risk factor effect ($p=0.027$) and a trend for time x metabolic syndrome risk factor effect ($p=0.06$) for HOMA. No overall time ($p=0.85$) or time x diet effect ($p=0.33$) was observed. When analyzed by delta change from baseline, the MS group tended to experience a *decrease* in HOMA (-0.29 ± 4.72) compared to an *increase* in the AH group ($+0.33 \pm 2.0$, $p=0.06$), however these values were not significantly different from baseline, nor were the ten-week values significantly different from one another. Post hoc analysis utilizing calculations on delta change from baseline demonstrate the HC-MS group was the only group to experience a significant change from baseline (-1.17 ± 7.2 , $p=0.030$), a significantly greater decrease in HOMA than HP-MS ($+0.25 \pm 1.9$, $p=0.039$) and HC-AH ($+0.65 \pm 3.0$, $p=0.008$). Figures 4.15 and 4.16 depict the delta changes for the glucose homeostasis parameters over the ten-week period. Based on these findings, hypothesis H_7 is accepted, which indicated there would be statistically significant differences in the markers of glucose homeostasis as a result of diet intervention and/or the presence of metabolic syndrome risk status.

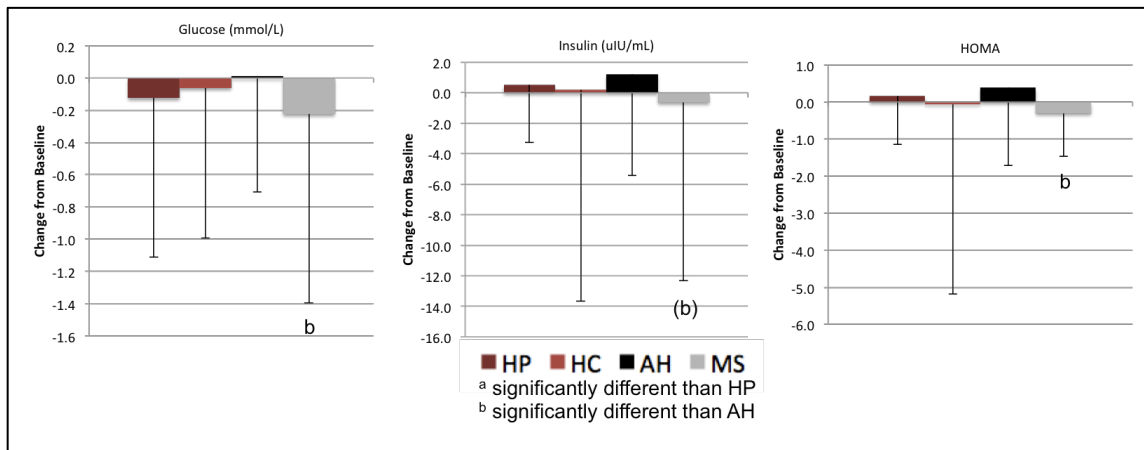


Figure 4.15: Delta Changes in Glucose Homeostasis Parameters, by Diet and Metabolic Syndrome Status

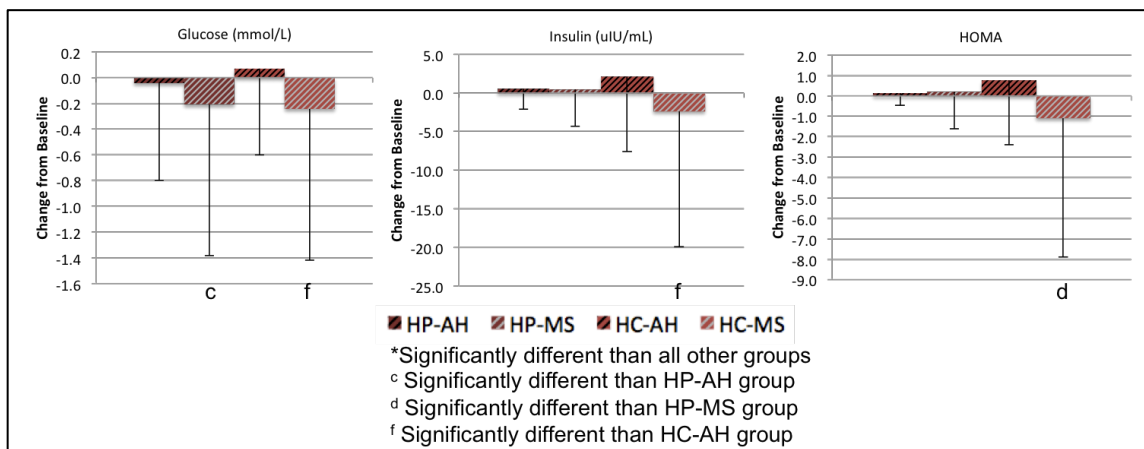


Figure 4.16: Delta Changes in Glucose Homeostasis Parameters, by Group-Combinations

Fitness Parameters

Table 4.16 depicts the time x diet x metabolic syndrome risk factor analysis for fitness parameters including maximum strength and lift volume for both bench press and leg press, as well as peak VO₂. The bench press and leg press variables were analyzed together utilizing a three-way MANOVA using a subset of 596 participants, while peak VO₂ was analyzed individually using a three-way ANOVA with the entire 663 participants.

The three-way MANOVA on strength and lift volume parameters revealed an overall time (Wilks' Lambda $p < 0.001$) effect. No significant time x diet (Wilks' Lambda $p = 0.39$), time x metabolic syndrome risk factor (Wilks' Lambda $p = 0.94$), or time x diet x metabolic syndrome risk factor (Wilks' Lambda $p = 0.26$) interactions were observed. Both bench press and leg press maximum strength demonstrated a significant time effect ($p < 0.001$), with bench press demonstrating an overall increase of 2.15 ± 4.99 1RM/kg body weight ($p < 0.001$), and leg press demonstrating an overall increase of 15.2 ± 28.6 1RM/kg body weight ($p < 0.001$). No strength or lift volume parameters experienced a diet or metabolic syndrome risk factor effect over time. While none of the variables experienced a significant three-way interaction, bench press lift volume did reveal a trend towards a time x diet x metabolic syndrome risk factor effect ($p = 0.060$). Review of the post hoc analysis based on calculated delta change from baseline revealed that both bench and leg press maximum strength experienced a significant change from baseline for all of the group-combinations, but no significant differences between group-combinations.

Table 4.16: Fitness Parameters After Ten Weeks of Exercise and Dietary Intervention Based on Metabolic Syndrome Status

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Bench Press Max Strength (1 RM / kg body weight)	HP-AH	31.0	± 7.7	33.3 ^g	± 7.7				T = 0.001
	HP-MS	30.9	± 7.5	32.5 ^g	± 7.1				D = 0.28
	HC-AH	29.2 ^c	± 7.9	31.5 ^{c,g}	± 8.1				M = 0.27
	HC-MS	31.0	± 7.6	33.3 ^g	± 8.0				D x M = 0.07
	HP	30.9	± 7.6	32.9 ^g	± 7.4	31.9	± 0.4	T x D = 0.36	
	HC	29.9	± 7.8	32.2 ^g	± 8.1	31.3	± 0.5	T x M = 0.48	
	AH	30.1	± 7.9	32.5 ^g	± 8.0	31.3	± 0.4	T x D x M = 0.45	
	MS	30.9	± 7.5	32.8 ^g	± 7.4	31.9	± 0.5		
Time	30.5	± 7.7	32.6 [*]	± 7.7					
Bench Press Lift Volume (kg)	HP-AH	174.7	± 68.5	182.8	± 74.4				T = 0.92
	HP-MS	186.5	± 90.3	181.1	± 87.3				D = 0.57
	HC-AH	177.8	± 78.7	168.6	± 77.0				M = 0.015
	HC-MS	193.4	± 92.6	198.4 ^f	± 81.3				D x M = 0.12
	HP	180.3	± 79.7	182.0	± 80.7	181.3	± 3.7	T x D = 0.64	
	HC	183.8	± 84.5	180.1	± 79.9	184.5	± 4.4	T x M = 0.97	
	AH	176.1	± 73.3	176.1	± 75.9	176.0	± 3.7	T x D x M = 0.06	
	MS	189.1 ^b	± 91.1	187.7 ^b	± 85.3	189.9 [‡]	± 4.3		
Time	181.8	± 81.8	181.2	± 80.3					
Leg Press Max Strength (1 RM / kg body weight)	HP-AH	162.6	± 46.2	177.9 ^g	± 53.0				T = 0.001
	HP-MS	165.8	± 50.1	182.5 ^g	± 51.6				D = 0.16
	HC-AH	155.2	± 54.3	169.9 ^g	± 58.6				M = 0.17
	HC-MS	163.4	± 44.5	176.7 ^g	± 49.3				D x M = 0.67
	HP	164.1	± 48.1	180.1 ^g	± 52.3	172.2	± 2.7	T x D = 0.42	
	HC	158.4	± 50.8	172.5 ^g	± 55.2	166.3	± 3.2	T x M = 0.99	
	AH	159.1	± 50.2	174.1 ^g	± 55.7	166.4	± 2.7	T x D x M = 0.59	
	MS	164.9	± 48.0	180.3 ^g	± 50.7	172.1	± 3.2		
Time	161.7	± 49.3	176.8 [*]	± 53.6					
Leg Press Lift Volume (kg)	HP-AH	1782.7	± 935.0	1889.6	± 965.4				T = 0.19
	HP-MS	1864.7	± 1004.8	1980.8	± 1190.5				D = 0.004
	HC-AH	1639.6	± 933.3	1593.7 ^c	± 883.5				M = 0.18
	HC-MS	1711.2	± 744.2	1732.4 ^d	± 928.0				D x M = 0.90
	HP	1821.8	± 968.3	1933.1 ^g	± 1077.9	1879.5 [†]	± 46.5	T x D = 0.10	
	HC	1667.3	± 864.3	1647.4 ^a	± 901.7	1669.2	± 55.0	T x M = 0.61	
	AH	1715.6	± 935.6	1751.0	± 938.3	1726.4	± 46.9	T x D x M = 0.70	
	MS	1806.5	± 916.2	1886.6	± 1103.1	1822.3	± 54.7		
Time	1755.4	± 927.5	1810.3	± 1015.1					
Peak VO ₂ (mL/kg/min)	HP-AH	20.7	± 4.7	22.5 ^g	± 4.7				T = 0.001
	HP-MS	19.3 ^c	± 4.2	21.3 ^{c,g}	± 4.2				D = 0.024
	HC-AH	20.8	± 4.4	23.4 ^g	± 4.8				M = 0.002
	HC-MS	20.0	± 3.8	22.6 ^{d,g}	± 4.6				D x M = 0.49
	HP	20.0	± 4.5	21.9 ^g	± 4.5	20.9 [†]	± 0.22	T x D = 0.016	
	HC	20.5	± 4.2	23.1 ^{a,g}	± 4.7	21.7	± 0.25	T x M = 0.80	
	AH	20.7	± 4.6	22.9 ^g	± 4.8	21.8	± 0.21	T x D x M = 0.64	
	MS	19.6 ^b	± 4.1	21.8 ^{b,g}	± 4.4	20.8 [‡]	± 0.25		
Time	20.2	± 4.4	22.4 [*]	± 4.6					
HP = higher protein, HC = higher carbohydrate, AH = apparently healthy, MS = metabolic syndrome risk factor T = time effect, D = diet effect, M = metabolic syndrome risk factor effect, D x M = diet by metabolic syndrome risk factor effect, T x D = time by diet effect, T x M = time by metabolic syndrome risk factor effect, T x D x M = time by diet by metabolic syndrome risk factor effect. Strength values are means ± standard deviations (except group means are means ± standard error) from 189 participants in the HP-AH group, 182 in the HP-MS group, 169 in the HC-AH group, 123 in the HC-MS group, 371 in the HP total group, 292 in the HC total group, 358 in the AH group, 305 in the MS group, and 663 participants total. Peak VO ₂ values are means ± standard deviations (except group means are means ± standard error) from 198 participants in the HP-AH group, 173 in the HP-MS group, 179 in the HC-AH group, 113 in the HC-MS group, 371 in the HP total group, 292 in the HC total group, 377 in the AH group, 286 in the MS group, and 663 participants total. * Significantly different than baseline, p < 0.05 (univariate). † Significant diet effect, p < 0.05 (univariate). ‡ Significant metabolic syndrome effect, p < 0.05 (univariate). ^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than AH group, p < 0.05 (post hoc LSD). ^c Significantly different than HP-AH group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-MS group, p < 0.05 (post hoc LSD). ^e Significantly different than HC-AH group, p < 0.05 (post hoc LSD). ^f Significantly different than baseline, p < 0.05 (post hoc LSD).									

The three-way ANOVA for peak VO_2 revealed an overall time ($p<0.001$) and time x diet ($p=0.016$) effect. No significant time x metabolic syndrome risk factor ($p=0.80$) or time x diet x metabolic syndrome risk factor ($p=0.64$) interactions were observed. Overall, participants gained an average of 2.2 ± 3.3 ml/kg/min (time effect, $p<0.001$) in aerobic capacity over the ten-week protocol. The HC group experienced a significantly greater increase in peak aerobic capacity (HP $+1.9\pm3.0$, HC $+2.6\pm3.7$ ml/kg/min, $p=0.016$). No difference was seen overtime between the health status groups. Post hoc analyses using calculated delta change from baseline revealed that the four group-combinations (HP-AH, HP-MS, HC-AH, and HC-MS) demonstrated significant increases from baseline ($p<0.001$ for each). Additionally, the HC-AH group ($+2.6\pm3.5$ ml/kg/min) experienced a significantly greater increase in peak VO_2 than the HP-AH group ($+1.8\pm3.2$ ml/kg/min, $p=0.026$). Figures 4.17 and 4.18 depict the delta changes in peak VO_2 over the ten-week period. Due to the significant time x diet effect observed with peak aerobic capacity, hypothesis H_8 , which indicated there would not be statistically significant differences in fitness parameters as a result of diet intervention and/or the presence of metabolic syndrome risk status, must be rejected.

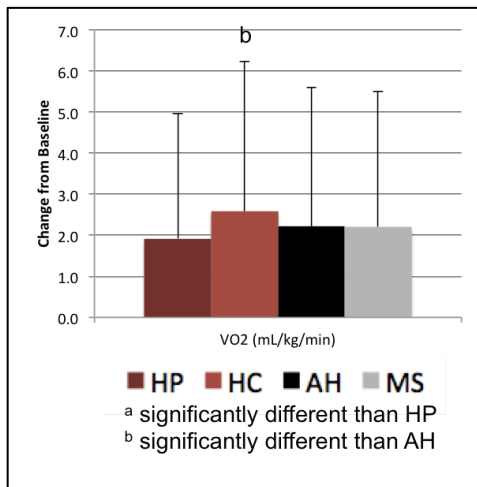


Figure 4.17: Delta Changes in Peak VO₂, by Diet and Metabolic Syndrome Status

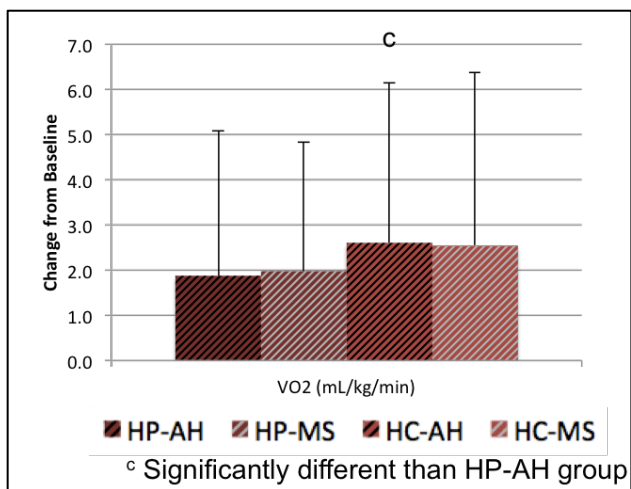


Figure 4.18: Delta Changes in Peak VO₂, by Group-Combination

Psychometric Analysis

The variables related to quality of life are broken down into two tables of analysis, based on the questionnaire titles: SF36 - Quality of Life and Body Image. The variables in each of these individual tables were collected in a questionnaire and run together in three-way MANOVAs.

Table 4.17 depicts the time x diet x metabolic syndrome risk factor analysis for SF36 – Quality of Life. The SF36 data was measured on a subset of 259 participants. The variables measured include physical function, role physical, bodily pain, general health, vital, social, role emotional, and mental health. The three-way MANOVA revealed an overall time effect (Wilks' Lambda $p < 0.001$), but no overall significant time x diet (Wilks' Lambda $p = 0.14$), time x metabolic syndrome risk factor (Wilks' Lambda $p = 0.35$), or time x diet x metabolic syndrome risk factor (Wilks' Lambda $p = 0.44$) effects.

Individual variables measured that experienced a time effect (including the overall effect and significance) include physical fitness ($+6.0 \pm 22.6$, $p < 0.001$), bodily pain ($+2.8 \pm 17.2$, $p = 0.010$), general health ($+3.9 \pm 11.9$, $p < 0.001$), vital ($+6.6 \pm 14.8$, $p < 0.001$), social ($+2.7 \pm 17.3$, $p = 0.017$), and mental health ($+8.0 \pm 15.0$, $p < 0.001$). The higher protein group tended to experience a greater increase in the social aspect over time than the higher carbohydrate group (HP $+4.4 \pm 15.5$, HC $+0.8 \pm 19.0$, $p = 0.089$). The apparently healthy group tended to experience an *increase* in the role physical aspect while the MS group experienced a decrease, though neither group experienced a significantly change from baseline (AH $+10.3 \pm 72.3$, MS -4.3 ± 62.9 , $p = 0.09$).

Table 4.17: Changes in SF36 - Quality of Life Inventory Values After Ten Weeks of Exercise and Dietary Intervention Based on Metabolic Syndrome Status

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Physical Functioning	HP-AH	79.0	± 31.1	86.1 ^g	± 27.4				T = 0.001
	HP-MS	71.5	± 27.6	75.3 ^c	± 19.6				D = 0.57
	HC-AH	77.6	± 23.8	82.9 ^g	± 21.7				M = 0.036
	HC-MS	74.6	± 17.1	82.7 ^g	± 18.1				D x M = 0.14
	HP	75.7	± 29.7	81.3 ^g	± 24.7	78.0	± 1.75		T x D = 0.64
	HC	76.4	± 21.2	82.8 ^g	± 20.2	79.4	± 1.86		T x M = 0.93
	AH	78.3	± 27.7	84.5 ^g	± 24.8	81.4	± 1.68		T x D x M = 0.24
	MS	72.9	± 23.3	78.7 ^{b,g}	± 19.2	76.0 [‡]	± 1.93		
	Time	76.0	± 26.0	82.0 [*]	± 22.7				
Role Physical	HP-AH	162.3	± 140.9	176.7	± 156.9				T = 0.44
	HP-MS	117.3 ^c	± 129.9	109.1 ^c	± 127.7				D = 0.001
	HC-AH	250.2 ^c	± 144.1	256.2 ^c	± 146.8				M = 0.06
	HC-MS	246.4 ^d	± 141.0	246.8 ^d	± 148.6				D x M = 0.13
	HP	142.2	± 137.5	146.4	± 148.0	141.3 [†]	± 11.30		T x D = 0.99
	HC	248.6 ^a	± 142.3	252.3 ^a	± 147.1	249.9	± 12.01		T x M = 0.09
	AH	205.2	± 148.7	215.5	± 156.8	211.4	± 10.82		T x D x M = 0.30
	MS	176.2	± 149.2	171.9 ^b	± 153.4	179.9	± 12.44		
	Time	192.6	± 149.4	196.6	± 156.5				
Bodily Pain	HP-AH	59.9	± 19.1	63.8 ^g	± 20.5				T = 0.010
	HP-MS	58.1	± 18.9	62.3 ^g	± 21.1				D = 0.48
	HC-AH	61.9	± 21.8	63.7	± 20.8				M = 0.65
	HC-MS	62.1	± 21.1	62.8	± 22.9				D x M = 0.78
	HP	59.1	± 19.0	63.1 ^g	± 20.7	61.0	± 1.54		T x D = 0.16
	HC	62.0	± 21.4	63.3	± 21.6	62.6	± 1.64		T x M = 0.85
	AH	60.9	± 20.4	63.8 ^g	± 20.6	62.3	± 1.47		T x D x M = 0.76
	MS	59.9	± 19.9	62.5	± 21.9	61.3	± 1.69		
	Time	60.5	± 20.2	63.2 [*]	± 21.1				
General Health	HP-AH	53.5	± 28.0	59.1 ^g	± 28.5				T = 0.001
	HP-MS	46.5	± 29.1	49.5 ^{c,g}	± 28.1				D = 0.001
	HC-AH	66.5 ^c	± 21.8	69.7 ^{c,g}	± 21.2				M = 0.054
	HC-MS	63.2 ^d	± 23.2	66.5 ^{d,g}	± 23.5				D x M = 0.40
	HP	50.4	± 28.6	54.8 ^g	± 28.6	52.1 [†]	± 2.04		T x D = 0.48
	HC	65.1 ^a	± 22.3	68.4 ^{a,g}	± 22.1	66.5	± 2.17		T x M = 0.38
	AH	59.8	± 25.9	64.3 ^g	± 25.7	62.2	± 1.95		T x D x M = 0.35
	MS	54.1	± 27.7	57.3 ^{b,g}	± 27.4	56.4 [‡]	± 2.24		
	Time	57.3	± 26.8	61.2 [*]	± 26.6				
Vital	HP-AH	39.6	± 21.8	48.7 ^g	± 27.5				T = 0.001
	HP-MS	43.3	± 64.7	49.7 ^g	± 64.4				D = 0.08
	HC-AH	51.3	± 17.7	55.5 ^g	± 16.7				M = 0.89
	HC-MS	49.0	± 19.6	55.6 ^g	± 21.0				D x M = 0.69
	HP	41.3	± 46.1	49.1 ^g	± 47.5	45.3	± 2.91		T x D = 0.18
	HC	50.4 ^a	± 18.5	55.5 ^g	± 18.6	52.9	± 3.09		T x M = 0.93
	AH	45.3	± 20.7	52.0 ^g	± 23.1	48.8	± 2.78		T x D x M = 0.15
	MS	45.9	± 49.4	52.4 ^g	± 49.5	49.4	± 3.20		
	Time	45.6	± 36.0	52.2 [*]	± 36.8				

Table 4.17: Continued

Variable	Group	Baseline	10 Weeks	Group (SEM)	P-level
Social Functioning	HP-AH	48.4 ± 26.0	54.8 ^s ± 26.6		T = 0.017
	HP-MS	43.6 ± 25.4	45.6 ^c ± 26.2		D = 0.001
	HC-AH	58.4 ^c ± 24.0	59.9 ± 22.7		M = 0.31
	HC-MS	60.4 ^d ± 26.3	60.4 ^d ± 27.0		D x M = 0.15
	HP	46.2 ± 25.7	50.7 ^s ± 26.7	48.1 [†] ± 1.95	T x D = 0.09
	HC	59.3 ^a ± 24.9	60.1 ^a ± 24.5	59.8 ± 2.08	T x M = 0.15
	AH	53.3 ± 25.5	57.3 ^s ± 24.8	55.4 ± 1.87	T x D x M = 0.49
	MS	51.3 ± 27.0	52.3 ± 27.5	52.5 ± 2.15	
	Time	52.4 ± 26.1	55.2 [*] ± 26.1		
Role Emotional	HP-AH	227.9 ± 127.3	255.0 ± 143.5		T = 0.44
	HP-MS	206.4 ± 130.4	218.3 ± 135.3		D = 0.001
	HC-AH	303.3 ^c ± 227.6	281.8 ± 136.2		M = 0.42
	HC-MS	292.0 ^d ± 121.8	300.0 ^d ± 131.0		D x M = 0.31
	HP	218.3 ± 128.7	238.6 ± 140.6	226.9 [†] ± 10.85	T x D = 0.11
	HC	298.6 ^a ± 190.3	289.4 ^a ± 133.9	294.3 ± 11.52	T x M = 0.66
	AH	264.7 ± 186.5	268.0 ± 140.2	267.0 ± 10.39	T x D x M = 0.17
	MS	245.4 ± 133.1	255.6 ± 138.9	254.2 ± 11.94	
	Time	256.3 ± 165.6	262.7 ± 139.6		
Mental Health	HP-AH	57.9 ± 15.4	67.3 ^s ± 15.3		T = 0.001
	HP-MS	54.9 ± 15.6	62.4 ^{c,g} ± 15.1		D = 0.86
	HC-AH	61.6 ± 14.6	66.5 ^s ± 12.6		M = 0.38
	HC-MS	58.3 ± 13.9	69.2 ^{d,g} ± 11.4		D x M = 0.99
	HP	56.5 ± 15.5	65.1 ^s ± 15.3	60.6 ± 1.01	T x D = 0.86
	HC	60.3 ^a ± 14.4	67.6 ^s ± 12.1	63.9 ± 1.07	T x M = 0.49
	AH	59.7 ± 15.1	66.9 ^s ± 14.0	63.3 ± 0.96	T x D x M = 0.70
	MS	56.4 ± 14.9	65.5 ^s ± 13.9	61.2 ± 1.11	
	Time	58.3 ± 15.1	66.3 [*] ± 13.9		

HP = higher protein, HC = higher carbohydrate, AH = apparently healthy, MS = metabolic syndrome risk factor, T = time effect, D = diet effect, M = metabolic syndrome risk factor effect, D x M = diet by metabolic syndrome risk factor effect, T x D = time by diet effect, T x M = time by metabolic syndrome risk factor effect, T x D x M = time by diet by metabolic syndrome risk factor effect.

Values are means ± standard deviations (except group means are means ± standard error) from 83 participants in the HP-AH group, 69 in the HP-MS group, 77 in the HC-AH group, 60 in the HC-MS group, 152 in the HP total group, 137 in the HC total group, 160 in the AH group, 129 in the MS group, and 289 participants total.

* Significantly different than baseline, $p < 0.05$ (univariate). † Significant diet effect, $p < 0.05$ (univariate). ‡ Significant metabolic syndrome effect, $p < 0.05$ (univariate). ^a Significantly different than HP group, $p < 0.05$ (post hoc LSD). ^b Significantly different than AH group, $p < 0.05$ (post hoc LSD). ^c Significantly different than HP-AH group, $p < 0.05$ (post hoc LSD). ^d Significantly different than HP-MS group, $p < 0.05$ (post hoc LSD). ^e Significantly different than baseline, $p < 0.05$ (post hoc LSD).

The mental health aspect was the only variable to experience a significant time x diet x metabolic syndrome risk factor effect ($p=0.028$). Post hoc analysis on mental health data expressed in delta changes from baseline revealed the HC-AH group had the least significant increase ($+4.9 \pm 12.5$, $p=0.051$) compared to all of the other group-combinations.

Table 4.18 depicts the time x diet x metabolic syndrome risk factor analysis pertaining to the Body Image Questionnaire. Body Image data was collected on a

subset of 451 participants. The variables measured include appearance evaluation, appearance orientation, body area satisfaction, overweight preoccupation, self-classified weight, Rosenberg self esteem, and social physique anxiety. The three-way MANOVA revealed an overall time effect (Wilks' Lambda $p < 0.001$), but no overall significant time x diet (Wilks' Lambda $p = 0.62$), time x metabolic syndrome risk factor (Wilks' Lambda $p = 0.40$), or time x diet x metabolic syndrome risk factor (Wilks' Lambda $p = 0.36$) effects.

Individual variables measured that experienced a time effect (including the overall effect and significance) include appearance evaluation ($+0.4 \pm 0.6$, $p < 0.001$), body area satisfaction ($+0.3 \pm 0.4$, $p < 0.001$), overweight preoccupation ($+0.5 \pm 0.7$, $p < 0.001$), self-classified weight (-0.1 ± 0.8 , $p = 0.007$), and Rosenberg self esteem ($+0.4 \pm 3.3$, $p = 0.005$). No Body Image variables experienced a diet effect over time. The metabolic syndrome group experienced a tendency for a greater decrease for self-classified weight over the ten-week protocol (AH -0.03 ± 0.8 , MS -0.2 ± 0.8 , $p = 0.089$). The only variable to experience a significant time x diet x metabolic syndrome risk factor interaction was body area satisfaction ($p = 0.044$). Post hoc analysis using delta calculations revealed a significant increase from baseline in body area satisfaction for all four group-combinations, and found HC-AH to have a significantly greater increase ($+0.34 \pm 0.48$) in body area satisfaction than all other group-combinations ($p = 0.037$).

Table 4.18: Changes in Body Image Evaluation After Ten Weeks of Exercise and Dietary Intervention Based on Metabolic Syndrome Status

Variable	Group	Baseline	10 Weeks	Group (SEM)	P-level
Appearance Evaluation	HP-AH	2.4 ± 0.7	2.8 ^g ± 0.7		T = 0.001
	HP-MS	2.4 ± 0.6	2.7 ^g ± 0.6		D = 0.57
	HC-AH	2.5 ± 0.7	2.8 ^g ± 0.7		M = 0.29
	HC-MS	2.4 ± 0.6	2.7 ^g ± 0.7		D x M = 0.53
	HP	2.4 ± 0.6	2.7 ^g ± 0.7	2.6 ± 0.04	T x D = 0.53
	HC	2.4 ± 0.6	2.8 ^g ± 0.7	2.6 ± 0.04	T x M = 0.51
	AH	2.4 ± 0.7	2.8 ^g ± 0.7	2.6 ± 0.04	T x D x M = 0.57
	MS	2.4 ± 0.6	2.7 ^g ± 0.6	2.6 ± 0.04	
Appearance Orientation	Time	2.4 ± 0.6	2.8* ± 0.7		
	HP-AH	4.1 ± 0.8	4.1 ± 0.9		T = 0.32
	HP-MS	4.1 ± 0.9	4.1 ± 0.9		D = 0.008
	HC-AH	4.0 ± 0.8	3.9 ± 0.9		M = 0.58
	HC-MS	3.9 ^d ± 0.8	3.8 ^d ± 0.8		D x M = 0.34
	HP	4.1 ± 0.9	4.1 ± 0.9	4.1† ± 0.05	T x D = 0.53
	HC	3.9 ^a ± 0.8	3.9 ^a ± 0.9	3.9 ± 0.06	T x M = 0.48
	AH	4.1 ± 0.8	4.0 ± 0.9	4.0 ± 0.05	T x D x M = 0.53
Body Area Satisfaction	MS	4.0 ± 0.9	4.0 ± 0.9	4.0 ± 0.06	
	Time	4.1 ± 0.8	4.0 ± 0.9		
	HP-AH	2.1 ± 0.7	2.3 ^g ± 0.8		T = 0.001
	HP-MS	1.9 ^c ± 0.8	2.2 ^g ± 0.9		D = 0.002
	HC-AH	2.3 ± 0.7	2.6 ^{c,g} ± 0.8		M = 0.046
	HC-MS	2.2 ^d ± 0.7	2.5 ^g ± 0.8		D x M = 0.73
	HP	2.0 ± 0.8	2.3 ^g ± 0.8	2.1† ± 0.05	T x D = 0.32
	HC	2.2 ^a ± 0.7	2.5 ^{a,g} ± 0.8	2.4 ± 0.06	T x M = 0.49
Overweight Preoccupation	AH	2.2 ± 0.7	2.5 ^g ± 0.8	2.3 ± 0.05	T x D x M = 0.044
	MS	2.0 ^b ± 0.7	2.3 ^g ± 0.9	2.2‡ ± 0.06	
	Time	2.1 ± 0.7	2.4* ± 0.8		
	HP-AH	2.8 ± 0.8	3.4 ^g ± 0.7		T = 0.001
	HP-MS	2.9 ± 0.6	3.4 ^g ± 0.7		D = 0.16
	HC-AH	2.9 ± 0.8	3.4 ^g ± 0.7		M = 0.37
	HC-MS	2.8 ± 0.7	3.2 ^{d,f,g} ± 0.7		D x M = 0.047
	HP	2.9 ± 0.7	3.4 ^g ± 0.7	3.1 ± 0.04	T x D = 0.11
Self-Classified Weight	HC	2.9 ± 0.7	3.3 ^{a,g} ± 0.7	3.1 ± 0.05	T x M = 0.77
	AH	2.9 ± 0.8	3.4 ^g ± 0.7	3.1 ± 0.04	T x D x M = 0.46
	MS	2.8 ± 0.6	3.3 ^g ± 0.7	3.1 ± 0.05	
	Time	2.9 ± 0.7	3.3* ± 0.7		
	HP-AH	4.2 ± 0.8	4.2 ± 0.6		T = 0.007
	HP-MS	4.3 ± 0.7	4.1 ^g ± 0.7		D = 0.11
	HC-AH	4.1 ± 0.7	4.0 ^c ± 0.7		M = 0.16
	HC-MS	4.3 ± 0.7	4.1 ± 0.8		D x M = 0.38
Self-Classified Weight	HP	4.2 ± 0.7	4.2 ± 0.7	4.2 ± 0.04	T x D = 0.62
	HC	4.2 ± 0.7	4.0 ^g ± 0.7	4.1 ± 0.04	T x M = 0.09
	AH	4.1 ± 0.7	4.1 ± 0.6	4.1 ± 0.04	T x D x M = 0.30
	MS	4.3 ^b ± 0.7	4.1 ^g ± 0.7	4.2 ± 0.04	
	Time	4.2 ± 0.7	4.1* ± 0.7		

Table 4.18: Continued

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Rosenberg Self Esteem	HP-AH	25.9	± 3.9	26.3	± 3.9				T = 0.005
	HP-MS	25.5	± 4.0	26.3 ^g	± 3.8				D = 0.55
	HC-AH	25.8	± 4.0	25.9	± 3.8				M = 0.62
	HC-MS	25.5	± 3.9	26.0	± 4.3				D x M = 0.93
	HP	25.7	± 3.9	26.3 ^g	± 3.8	26.0	± 0.22	T x D = 0.26	
	HC	25.7	± 3.9	25.9	± 4.0	25.8	± 0.26	T x M = 0.22	
	AH	25.9	± 4.0	26.1	± 3.9	26.0	± 0.22	T x D x M = 0.90	
	MS	25.5	± 3.9	26.2 ^g	± 4.0	25.8	± 0.27		
	Time	25.7	± 3.9	26.2 [*]	± 3.9				
Social Physique Anxiety	HP-AH	31.1	± 6.4	31.1	± 6.6				T = 0.60
	HP-MS	31.4	± 6.1	31.7	± 5.8				D = 0.86
	HC-AH	31.1	± 6.2	31.0	± 5.1				M = 0.38
	HC-MS	31.2	± 5.6	31.8	± 7.5				D x M = 0.99
	HP	31.3	± 6.3	31.4	± 6.2	31.3	± 0.32	T x D = 0.86	
	HC	31.1	± 6.0	31.3	± 6.1	31.2	± 0.38	T x M = 0.49	
	AH	31.1	± 6.3	31.0	± 6.0	31.1	± 0.32	T x D x M = 0.70	
	MS	31.3	± 5.9	31.7	± 6.5	31.5	± 0.38		
	Time	31.2	± 6.1	31.3	± 6.2				
HP = higher protein, HC = higher carbohydrate, AH = apparently healthy, MS = metabolic syndrome risk factor, T = time effect, D = diet effect, M = metabolic syndrome risk factor effect, D x M = diet by metabolic syndrome risk factor effect, T x D = time by diet effect, T x M = time by metabolic syndrome risk factor effect, T x D x M = time by diet by metabolic syndrome risk factor effect. Values are means ± standard deviations (except group means are means ± standard error) from 139 participants in the HP-AH group, 117 in the HP-MS group, 117 in the HC-AH group, 78 in the HC-MS group, 256 in the HP total group, 195 in the HC total group, 256 in the AH group, 195 in the MS group, and 451 participants total. * Significantly different than baseline, p < 0.05 (univariate). † Significant diet effect, p < 0.05 (univariate). ‡ Significant metabolic syndrome effect, p < 0.05 (univariate). ^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than AH group, p < 0.05 (post hoc LSD). ^c Significantly different than HP-AH group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-MS group, p < 0.05 (post hoc LSD). ^e Significantly different than HC-AH group, p < 0.05 (post hoc LSD). ^f Significantly different than baseline, p < 0.05 (post hoc LSD).									

Based on the MANOVA univariate tests, revealing a significant time x diet x metabolic syndrome risk factor interaction for mental health (SF36), and body area satisfaction (Body Image), hypothesis H₉, which stated there would be no significant differences observed in psychometric parameters as a result of diet intervention and/or the presence of the metabolic syndrome health status, is rejected.

CHANGES IN METABOLIC SYNDROME STATUS

Table 4.19 depicts the N-sizes and percentages of participants who were identified with each metabolic syndrome risk factor both at baseline and at the end of the ten-week protocol. By the end of the study, the prevalence of each risk factor for the

entire study population changed as follows: waist circumference -13%, glucose -4.2%, HDL +8.9%, blood pressure -7%, and triglycerides -4.4%.

Table 4.20 depicts the number of risk factors experienced by each participant both at baseline and the end of the ten-week protocol. Overall, the prevalence of metabolic syndrome for the study population dropped from 43% to 38%, a decrease of 5.6% of the population.

Table 4.19: Baseline and Ten-Week Frequencies per Metabolic Syndrome Risk Factor

Risk Factor	Baseline			10 Weeks		
	HP	HC	TTL	HP	HC	TTL
Waist Circumference	316 (85%)	215 (74%)	531 (80%)	269 (73%) -12%	176 (60%) -14%	445 (67%) -13%
Glucose	157 (42%)	116 (40%)	273 (41%)	140 (38%) -4%	105 (36%) -4%	245 (37%) -4%
HDL Cholesterol	151 (41%)	112 (38%)	263 (40%)	188 (51%) +10%	134 (46%) +8%	322 (49%) +9%
Blood Pressure	148 (40%)	96 (33%)	244 (37%)	121 (33%) -7%	75 (26%) -7%	196 (30%) -7%
Triglycerides	124 (33%)	96 (33%)	220 (33%)	109 (29%) -4%	82 (28%) -4%	191 (29%) -4%
HP = higher protein; HC = higher carbohydrate; TTL = total n-size. Percentages are based on the n-size for HP (n=371), HC (n=292), and total (n=663)						

Table 4.20: Ten-Week Changes in the Number of Risk Factors per Diet Group

Number of Risk Factors	Baseline			10 Weeks		
	HP	HC	Total	HP	HC	Total
0	18	26	44	38	43	81
1	69	61	130	66	66	132
2	111	92	203	119	82	201
3	105	68	173	83	68	151
4	50	39	89	51	27	78
5	18	6	24	14	6	20
HC, higher carbohydrate; HP, higher protein, N-size for HP (n=371), HC (n=292), and total (n=663)						

ANALYSIS OF INDIVIDUAL METABOLIC SYNDROME RISK FACTORS

In addition to the retrospective stratification of participants based on metabolic syndrome status, the study population was also retrospectively stratified based on each metabolic syndrome risk factor individually. Groups classified as having the individual metabolic syndrome risk factor included high waist circumference, high triglycerides, low HDL cholesterol, high blood pressure, and high fasting glucose. Each of the five risk factor stratifications were analyzed independently with diet composition (higher protein versus higher carbohydrate consumption) in order to differentiate the beneficial effects of diet on each risk factor individually.

ANALYSIS OF WAIST CIRCUMFERENCE AS A RISK FACTOR

The metabolic syndrome waist circumference risk factor for women has been defined as a waist circumference value greater than 88 cm. Participants have been identified as either low waist circumference (LWC; ≤ 88 cm, N=132), or high waist circumference (HWC; > 88 cm, N=531).

Energy Intake

Table 4.21 depicts the time x diet x waist circumference risk factor MANOVA for total energy intake in kcals/kg/day as well as macronutrient intake in g/kg/day. The three-way MANOVA revealed overall time (Wilks' Lambda $p < 0.001$), time x diet (Wilks' Lambda $p < 0.001$), time x waist circumference risk factor (Wilks' Lambda $p = 0.002$), and time x diet x waist circumference risk factor (Wilks' Lambda $p = 0.045$) effects. Post hoc analysis on the ten-week values was utilized to determine the following:

Table 4.21: Dietary Intake Before and After Ten Weeks of Exercise and Dietary Intervention Based on Waist Circumference Risk Factor Status

Variable	Group	Baseline			10 Weeks			Group (SEM)			P-level
Energy Intake (kcal/kg/day)	HP-LWC	22.6	±	6.0	20.2 ^g	±	5.2				T = 0.001
	HP-HWC	20.0 ^c	±	5.6	15.2 ^{cg}	±	4.8				D = 0.53
	HC-LWC	22.0	±	5.1	19.8 ^g	±	5.1				WC = 0.001
	HC-HWC	20.7	±	7.0	16.5 ^{dfe}	±	4.3				D x WC = 0.08
	HP	20.4	±	5.8	15.9 ^g	±	5.2	19.5	±	0.3	T x D = 0.47
	HC	21.0	±	6.6	17.4 ^g	±	4.7	19.8	±	0.3	T x WC = 0.001
	LWC	22.3	±	5.5	19.9 ^g	±	5.1	21.2	±	0.4	T x D x WC = 0.69
	HWC	20.3 ^b	±	6.2	15.7 ^{bg}	±	4.6	18.1 [‡]	±	0.2	
	Time	20.7	±	6.1	16.6 [*]	±	5.0				
Protein Intake (g/kg/day)	HP-LWC	0.95	±	0.2	1.46 ^g	±	0.6				T = 0.001
	HP-HWC	0.81 ^c	±	0.2	1.09 ^{cg}	±	0.5				D = 0.001
	HC-LWC	0.88	±	0.2	0.85 ^c	±	0.2				WC = 0.001
	HC-HWC	0.81 ^f	±	0.2	0.76 ^d	±	0.3				D x WC = 0.001
	HP	0.83	±	0.2	1.14 ^g	±	0.5	1.08 [†]	±	0.0	T x D = 0.001
	HC	0.83	±	0.2	0.78 ^a	±	0.3	0.83	±	0.0	T x WC = 0.003
	LWC	0.91	±	0.2	1.10 ^g	±	0.5	1.04	±	0.0	T x D x WC = 0.009
	HWC	0.81 ^b	±	0.2	0.95 ^{bg}	±	0.4	0.87 [‡]	±	0.0	
	Time	0.83	±	0.2	0.98 [*]	±	0.4				
Carbohydrate Intake (g/kg/day)	HP-LWC	2.65	±	0.9	1.80 ^g	±	0.8				T = 0.001
	HP-HWC	2.36 ^c	±	0.9	1.35 ^{cg}	±	0.6				D = 0.001
	HC-LWC	2.66	±	0.8	2.59 ^c	±	0.8				WC = 0.001
	HC-HWC	2.47	±	1.0	2.07 ^{dfe}	±	0.6				D x WC = 0.88
	HP	2.40	±	0.9	1.41 ^g	±	0.7	2.04 [†]	±	0.0	T x D = 0.001
	HC	2.52	±	0.9	2.20 ^{ag}	±	0.7	2.45	±	0.0	T x WC = 0.008
	LWC	2.66	±	0.8	2.26 ^g	±	0.9	2.43	±	0.1	T x D x WC = 0.35
	HWC	2.40 ^b	±	0.9	1.64 ^{bg}	±	0.7	2.06 [‡]	±	0.0	
	Time	2.46	±	0.9	1.76 [*]	±	0.8				
Fat Intake (g/kg/day)	HP-LWC	0.91	±	0.3	0.79 ^g	±	0.3				T = 0.001
	HP-HWC	0.82 ^c	±	0.3	0.60 ^{cg}	±	0.3				D = 0.048
	HC-LWC	0.87	±	0.2	0.67 ^{cg}	±	0.2				WC = 0.001
	HC-HWC	0.84	±	0.4	0.58 ^{fg}	±	0.2				D x WC = 0.044
	HP	0.83	±	0.3	0.63 ^g	±	0.3	0.78 [†]	±	0.0	T x D = 0.057
	HC	0.85	±	0.3	0.60 ^{ag}	±	0.2	0.74	±	0.0	T x WC = 0.028
	LWC	0.89	±	0.3	0.72 ^g	±	0.3	0.81	±	0.0	T x D x WC = 0.55
	HWC	0.83 ^b	±	0.3	0.59 ^{bg}	±	0.2	0.71 [‡]	±	0.0	
	Time	0.84	±	0.3	0.62 [*]	±	0.2				

HP = higher protein, HC = higher carbohydrate, LWC = low waist circumference, HWC = high waist circumference, T = time effect, D = diet effect, WC = waist circumference risk factor effect, D x WC = diet by waist circumference risk factor effect, T x D = time by diet effect, T x WC = time by waist circumference risk factor effect, T x D x WC = time by diet by waist circumference risk factor effect.

Values are means ± standard deviations (except group means are means ± standard error) from 55 participants in the HP-LWC group, 316 in the HP-HWC group, 77 in the HC-LWC group, 215 in the HC-HWC group, 371 in the HP total group, 292 in the HC total group, 132 in the LWC group, 531 in the HWC group, and 663 participants total.

* Significantly different than baseline, $p < 0.05$ (univariate). † Significant diet effect, $p < 0.05$ (univariate). ‡ Significant waist circumference effect, $p < 0.05$ (univariate). ^a Significantly different than HP group, $p < 0.05$ (post hoc LSD). ^b Significantly different than LWC group, $p < 0.05$ (post hoc LSD). ^c Significantly different than HP-LWC group, $p < 0.05$ (post hoc LSD). ^d Significantly different than HP-HWC group, $p < 0.05$ (post hoc LSD). ^e Significantly different than HC-LWC group, $p < 0.05$ (post hoc LSD). ^f Significantly different than baseline, $p < 0.05$ (post hoc LSD).

While there was no significant difference between the diet groups for energy intake, participants in the LWC group consumed significantly more calories relative to body weight (LWC 19.9 ± 5.1 , HWC 15.7 ± 5.1 kcal/kg/d, $p < 0.001$). Further review of the individual group-combinations revealed HP-LWC consumed significantly more calories than HP-HWC ($p = 0.004$), HC-LWC consumed significantly more calories than HC-HWC ($p < 0.001$), and HC-HWC consumed significantly more calories than HP-HWC ($p = 0.001$). Participants in the HP group and the LWC group consumed significantly more protein relative to body weight (HP 1.14 ± 0.5 , HC 0.78 ± 0.3 g/kg/d, $p < 0.001$) (LWC 1.10 ± 0.5 , HWC 0.95 ± 0.4 g/kg/d, $p < 0.001$). Specifically, the HP-LWC group consumed significantly more protein than HP-HWC ($p < 0.001$) and HC-LWC ($p < 0.001$), and HP-HWC consumed significantly more protein than HC-HWC ($p < 0.001$). The HC group and the LWC group consumed significantly more carbohydrate relative to body weight (HP 1.41 ± 0.7 , HC 2.20 ± 0.7 g/kg/d, $p < 0.001$), (LWC 2.26 ± 0.9 , HWC 1.64 ± 0.7 g/kg/d, $p < 0.001$). All group-combinations were significantly different from one another, with the HC-LWC group consuming the greatest amount of carbohydrate ($p < 0.001$). Finally, the HP group and the LWC group consumed significantly more fat relative to body weight (HP 0.63 ± 0.3 , HC 0.60 ± 0.2 g/kg/d, $p = 0.002$), (LWC 0.72 ± 0.3 , HWC 0.59 ± 0.2 g/kg/d, $p < 0.001$), with the HP-LWC group consuming a significantly greater amount than HP-HWC ($p < 0.001$) and HC-LWC ($p = 0.003$), and HC-LWC consuming a significantly greater amount than HC-HWC ($p = 0.005$).

Overall Analysis

Table 4.22 represents the Greenhouse-Geisser univariate and (when applicable) Wilks' Lambda multivariate p-levels for each of the variables and groups measured and analyzed by the combined diet and waist circumference risk factor interactions (time, diet, waist circumference risk factor, time x diet, time x waist circumference risk factor, diet x waist circumference risk factor, and time x diet x waist circumference risk factor). The significant findings per variable are discussed below. When a significant three-way interaction was observed, the significant group differences revealed in the post hoc LSD pairwise comparisons analysis are also indicated. Each corresponding hypothesis is also evaluated.

Body Composition

A three-way MANOVA on DEXA variables revealed an overall time (Wilks' Lambda $p < 0.001$) and a trend in time x diet effect (Wilks' Lambda $p = 0.063$). Analysis of MANOVA univariate tests revealed a greater decrease with the higher protein diet for scanned mass (HP -3.9 ± 3.5 , HC -3.0 ± 3.5 kg, $p = 0.032$), fat mass (HP -3.1 ± 2.7 , HC -2.4 ± 2.8 kg, $p = 0.007$), and body fat percentage (HP -1.6 ± 1.9 , HC $-1.4 \pm 2.0\%$, $p = 0.010$). A significant time x waist circumference risk factor interaction (Wilks' Lambda $p = 0.006$) showed a greater decrease in the high waist circumference group for both scanned mass (LWC -2.6 ± 3.5 , HWC -3.7 ± 3.5 kg, $p = 0.003$) and fat mass (LWC -2.1 ± 2.5 , HWC -3.0 ± 2.8 kg, $p = 0.006$). Additionally a time x diet x waist circumference risk factor trend (Wilks' Lambda $p = 0.078$) was observed, and the MANOVA univariate test for body fat showed significance ($p = 0.015$).

Table 4.22: Significance Levels for Waist Circumference Risk Factor Analysis per Variable

	Multivariate and Univariate P-Values							Post Hoc Significance							
	T	D	T*D	WC	T*WC	D*WC	T*D*WC	HP-LWC	HP-HWC	HC-LWC	HC-HWC	HP	HC	LWC	HWC
Body Composition	0.001		0.063		0.006		0.078								
Scanned Mass	0.001	0.105	0.032	0.001	0.003	0.018	0.82	g	c,g	g	d,f,g	g	g	b, g	g
Fat Mass	0.001	0.21	0.007	0.001	0.006	0.006	0.434	g	c,g	g	d,f,g	g	g	b, g	g
Lean Mass	0.001	0.035	0.98	0.001	0.16	0.19	0.14		c,g	g	d,f,g	g	a,g	b, g	g
Body Fat	0.001	0.43	0.010	0.001	0.78	0.007	0.015	g	c,g	c,g	d,f,g	g	g	b, g	g
Weight	0.001	0.12	0.004	0.001	0.001	0.020	0.60	g	c,g	g	d,f,g	g	g	b,g	g
Body Mass Index	0.001	0.042	0.004	0.001	0.001	0.034	0.61	g	c,g	g	d,f,g	g	g	b,g	g
Waist Circumference	0.001	0.045	0.27	0.001	0.001	0.053	0.62		c,g		f,g	g	a,g	b,g	g
Hip Circumference	0.001	0.25	0.12	0.001	0.15	0.029	0.234	g	c,g	g	d,f,g	g	g	b,g	g
Resting Energy Expenditure	0.001	0.23	0.35	0.001	0.44	0.046	0.97	g	c,g	g	d,f,g	g	g	b,g	g
Hemodynamic	0.001		0.78		0.035		0.77								
Resting Heart Rate	0.001	0.98	0.45	0.35	0.005	0.21	0.30		g		g	g	g		g
Systolic Blood Pressure	0.001	0.14	0.67	0.001	0.62	0.28	0.81		g	g	f,g	g	g	b,g	g
Diastolic Blood Pressure	0.001	0.011	0.46	0.001	0.55	0.88	0.80		c,g	g	d,f,g	g	a,g	b,g	g
Blood Lipids	0.001		0.70		0.30		0.83								
Total Cholesterol	0.001	0.27	0.35	0.62	0.25	0.021	0.77	g	g	g	g	g	g	g	g
LDL Cholesterol	0.001	0.33	0.45	0.57	0.14	0.009	0.69	g	g	g	f,g	g	g	g	g
HDL Cholesterol	0.001	0.76	0.88	0.001	0.98	0.88	0.87	g	c,g	g	f,g	g	g	b,g	g
Triglycerides	0.008	0.62	0.11	0.005	0.36	0.63	0.92		g		f	g		b	g
TC/HDL Ratio	0.38	0.66	0.50	0.001	0.14	0.150	0.89				f,g			b	g
Glucose	0.09	0.36	0.18	0.022	0.51	0.46	0.20		g			g		b	g
Insulin	0.20	0.035	0.80	0.94	0.023	0.12	0.28			g	d			g	
HOMA	0.36	0.055	0.71	0.95	0.031	0.08	0.23				d				
Maximum Strength	0.001		0.91		0.34		0.67								
Bench Press Max Strength	0.001	0.17	0.74	0.06	0.91	0.46	0.61	g	g	g	f,g	g	g	g	g
Bench Press Lift Volume	0.51	0.91	0.58	0.040	0.18	0.78	0.86								
Leg Press Max Strength	0.001	0.78	0.72	0.001	0.32	0.52	0.22	g	c,g	g	f,g	g	g	b,g	g
Leg Press Lift Volume	0.96	0.102	0.46	0.001	0.23	0.63	0.50		c,g		d,f			b	
Peak VO ₂	0.001	0.74	0.001	0.001	0.095	0.12	0.044	g	c,g	g	d,f,g	g	g	b,g	g

Table 4.22: Continued

	Multivariate and Univariate P-Values							Post Hoc Significance							
	T	D	T*D	WC	T*WC	D*WC	T*D*WC	HP-LWC	HP-HWC	HC-LWC	HC-HWC	HP	HC	LWC	HWC
SF36 Questionnaire	0.001		0.67		0.91		0.80								
Physical Function	0.001	0.701	0.90	0.095	0.65	0.35	0.91		g	g	g	g	g	g	g
Role Physical	0.63	0.001	0.82	0.85	0.44	0.045	0.60				d		a		
Bodily Pain	0.006	0.76	0.22	0.31	0.28	0.82	0.93		g			g		g	
General Health	0.001	0.010	0.48	0.020	0.92	0.044	0.96	g	c.g		d.g	g	a.g	b.g	g
Vital	0.001	0.19	0.48	0.63	0.72	0.79	0.31		g	g	g	g	g	g	g
Social	0.102	0.08	0.23	0.24	0.37	0.009	0.81		c.g		d				g
Role Emotional	0.47	0.003	0.28	0.09	0.55	0.17	0.33				d		a		
Mental Health	0.001	0.20	0.94	0.025	0.83	0.78	0.30	g	g	g	g	g	g	b.g	g
Body Image Questionnaire	0.001		0.95		0.26		0.49								
Appearance Evaluation	0.001	0.27	0.42	0.001	0.004	0.037	0.13	g	c.g	c.g	g	g	g	b.g	g
Appearance Orientation	0.16	0.24	0.90	0.20	0.56	0.25	0.43				d				
Body Area Satisfaction Scale	0.001	0.23	0.68	0.001	0.29	0.09	0.38	g	c.g	g	d.g	g	g	b.g	g
Overweight Preoccupation	0.001	0.95	0.31	0.15	0.70	0.25	0.80	g	g	g	g	g	g	g	g
Self Classified Weight	0.045	0.97	0.87	0.001	0.86	0.18	0.55		c		g			b	g
Rosenberg Self Esteem Scale	0.10	0.77	0.97	0.60	0.72	0.63	0.07		g						g
Social Physique Anxiety Scale	0.79	0.40	0.87	0.047	0.83	0.19	0.65								

T = time effect, D = diet effect, T x D = time by diet effect, WC = waist circumference risk factor effect, T x WC = time by waist circumference risk factor effect, D x WC = diet by waist circumference risk factor effect, T x D x WC = time by diet by waist circumference risk factor effect. HP = higher protein, HC = higher carbohydrate, LWC = low waist circumference (≤ 88 cm), HWC = high waist circumference (> 88 cm). N=663 participants unless stated otherwise as follows: REE N=633, Insulin and HOMA N=252, Strength N=596, SF36 N=289, and Body Image N=451. Group breakdowns are as follows unless otherwise stated: 55 participants in the HP-LWC group, 316 in the HP-HWC group, 77 in the HC-LWC group, 215 in the HC-HWC group, 371 in the HP total group, 292 in the HC total group, 132 in the LWC group, 531 in the HWC group, and 663 participants total; for REE: 47 participants in the HP-LWC group, 310 in the HP-HWC group, 67 in the HC-LWC group, 209 in the HC-HWC group, 357 in the HP total group, 276 in the HC total group, 114 in the LWC group, 519 in the HWC group, and 633 participants total; for insulin and HOMA: 25 participants in the HP-LWC group, 124 in the HP-HWC group, 40 in the HC-LWC group, 63 in the HC-HWC group, 149 in the HP total group, 103 in the HC total group, 65 in the LWC group, 187 in the HWC group, and 252 participants total; for strength: 41 participants in the HP-LWC group, 299 in the HP-HWC group, 56 in the HC-LWC group, 200 in the HC-HWC group, 340 in the HP total group, 256 in the HC total group, 97 in the LWC group, 499 in the HWC group, and 596 participants total; for SF36: 25 participants in the HP-LWC group, 127 in the HP-HWC group, 43 in the HC-LWC group, 94 in the HC-HWC group, 152 in the HP total group, 137 in the HC total group, 68 in the LWC group, 221 in the HWC group, and 289 participants total; and for Body Image: 37 participants in the HP-LWC group, 219 in the HP-HWC group, 60 in the HC-LWC group, 135 in the HC-HWC group, 256 in the HP total group, 195 in the HC total group, 97 in the LWC group, 354 in the HWC group, and 451 participants total. Wilks' Lambda Multivariate p-values for the group of variables are listed in bold. Greenhouse-Geisser Univariate p-values are listed for each variable included in the MANOVA (or ANOVA when indicated) analysis. Superscripts indicate significant differences between groups using Post Hoc LSD Pairwise Comparisons.

^a Significantly different than HP group, $p < 0.05$ (post hoc LSD). ^b Significantly different than HWC group, $p < 0.05$ (post hoc LSD). ^c Significantly different than HP-LWC group, $p < 0.05$ (post hoc LSD). ^d Significantly different than HP-HWC group, $p < 0.05$ (post hoc LSD). ^e Significantly different than HC-LWC group, $p < 0.05$ (post hoc LSD). ^f Significantly different than baseline, $p < 0.05$ (post hoc LSD).

Post hoc analysis of body composition data expressed in delta changes from baseline revealed the HP-LWC group demonstrated the greatest percent fat loss ($-1.9 \pm 2.3\%$, $p=0.044$).

Individual three-way ANOVAs were performed on weight, body mass index, waist circumference, and hip circumference. A significant time x diet effect indicated a greater loss in the higher protein group for both weight (HP -4.3 ± 3.6 , HC -3.2 ± 3.4 kg, $p=0.004$) and body mass index (HP -1.6 ± 1.3 , HC -1.2 ± 1.3 kg/m², $p=0.004$). No significant time x diet effect was observed for waist circumference ($p=0.272$) or hip circumference ($p=0.12$). Significant time x waist circumference risk factor effects were observed indicating a greater loss in the HWC group for weight (LWC -2.6 ± 2.9 , HWC -4.1 ± 3.6 kg, $p<0.001$), body mass index (LWC -1.0 ± 1.1 , HWC -1.5 ± 1.3 kg/m², $p<0.001$), and waist circumference (LWC -0.9 ± 4.2 , HWC -4.3 ± 5.9 cm, $p<0.001$). The time x waist circumference risk factor interaction for hip circumference was not significant ($p=0.15$). No significant time x diet x waist circumference risk factor interactions were observed for weight ($p=0.60$), body mass index ($p=0.61$), waist circumference ($p=0.62$), or hip circumference ($p=0.24$). Based on these findings, hypothesis H₁₀ is accepted, which indicated there would be statistically significant differences observed in changes in body composition as a result of diet intervention and/or the presence of the waist circumference metabolic syndrome risk factor.

Resting Energy Expenditure

The three-way ANOVA for resting energy expenditure and waist circumference risk factor revealed an overall time effect ($p<0.001$), but no significant interactions.

Therefore, hypothesis H_{11} is accepted, which stated there would be no statistically significant differences observed in resting energy expenditure as a result of diet intervention and/or the presence of the waist circumference metabolic syndrome risk factor.

Resting Hemodynamic Parameters

A three-way MANOVA on the resting hemodynamic parameters revealed an overall time (Wilks' Lambda $p < 0.001$) and time x waist circumference risk factor effect (Wilks' Lambda $p = 0.035$), with univariate significance observed specifically for resting heart rate ($p = 0.005$). This indicated a significantly greater decrease in heart rate for the HWC group over the ten-week protocol (LWC -0.82 ± 9.6 , HWC -3.6 ± 10.4 bpm, $p = 0.005$). No significant time x diet (Wilks' Lambda, $p = 0.78$) or time x diet x waist circumference risk factor (Wilks' Lambda $p = 0.77$) interactions were observed. Therefore, hypothesis H_{12} is rejected, which stated there would be no statistically significant differences observed in resting hemodynamic parameters as a result of diet intervention and/or the presence of the waist circumference metabolic syndrome risk factor.

Blood Lipids

While the three-way MANOVA for blood lipids revealed an overall time effect (Wilks' Lambda $p < 0.001$), no significant time x diet (Wilks' Lambda $p = 0.70$), time x waist circumference risk factor (Wilks' Lambda $p = 0.30$), or time x diet x waist circumference risk factor (Wilks' Lambda $p = 0.83$) interactions were observed. Based on these findings hypothesis H_{13} is rejected, which stated there would be statistically significant differences observed in blood lipids as a result of diet intervention, and

hypothesis H₁₄ is accepted, which stated there would be no statistically significant differences observed in blood lipids as a result of the presence of the waist circumference metabolic syndrome risk factor.

Glucose Homeostasis

No significant time effect or interactions were observed for the three-way ANOVA on glucose. While both insulin and HOMA variables did not experience significant time ($p=0.20$ and $p=0.36$) or time x diet interactions ($p=0.80$ and $p=0.71$ respectively), both variables demonstrated a significant time x waist circumference risk factor effect. The LWC group demonstrated a significantly greater *increases* in comparison to mild *decreases* in the HWC group (that was not significantly different from baseline) for both insulin (LWC $+2.59 \pm 9.7$, HWC -0.4 ± 9.0 uIU/mL, $p=0.023$) and HOMA (LWC $+0.8 \pm 3.3$, HWC -0.2 ± 3.4 , $p=0.031$). Based on the findings from this analysis hypothesis H₁₅ is accepted, which stated there would be statistically significant differences observed in markers of glucose homeostasis as a result of diet intervention and/or the presence of the waist circumference metabolic syndrome risk factor.

Fitness Parameters

The three-way MANOVA on maximum strength revealed an overall time effect (Wilks' Lambda $p<0.001$), but no significant interactions. However, an ANOVA on cardiorespiratory fitness, measured by peak VO₂, demonstrated significant effects with time, ($p<0.001$), time x diet ($p=0.001$) and time x diet x waist circumference risk factor ($p=0.044$), as well as a trend towards significance for time x waist circumference risk factor ($p=0.095$). Analysis of the MANOVA univariate tests revealed the HC group

experienced a significantly greater increase in peak VO_2 (HP $+1.9 \pm 3.0$, HC 2.6 ± 3.7 ml/kg/min, $p=0.001$), and the HWC group tended to experience a greater increase in peak VO_2 (LWC $+1.9 \pm 3.8$, HWC $+2.3 \pm 3.2$ ml/kg/min, $p=0.095$). Post hoc analysis on the time x diet x waist circumference risk factor using calculated delta change from baseline indicated that the HC-LWC group experienced the greatest gain in cardiorespiratory fitness ($+2.66 \pm 4.0$ ml/kg/min, $p=0.013$). Due to the findings regarding cardiorespiratory fitness, hypothesis H_{16} is rejected. This hypothesis stated there would be no statistically significant differences observed in fitness parameters as a result of diet intervention and/or the presence of the waist circumference metabolic syndrome risk factor.

Psychometric Analysis

While overall time effects (Wilks' Lambda $p<0.001$) were observed for both the SF36-Quality of Life Questionnaire and the Body Image Questionnaires, the three-way MANOVAs did not reveal any significant interaction effects for the waist circumference risk factor analysis. However review of the MANOVA univariate tests on the Body Image Questionnaire revealed a significant time x waist circumference risk factor interaction for appearance evaluation, indicating a significantly greater increase in the LWC group (LWC 0.50 ± 0.6 , HWC 0.31 ± 0.6 , $p=0.004$). The univariate time x diet x waist circumference risk factor for the Rosenberg Self Esteem Scale revealed a trend ($p=0.07$), with post hoc analysis on the calculated delta changes from baseline revealed HP-HWC tended to experience a greater increase compared to HC-HWC (HP-HWC $+0.74 \pm 3.3$, HC-HWC $+0.04 \pm 3.6$, $p=0.055$). Based on the time x waist circumference

risk factor interaction for appearance evaluation, hypothesis H_{17} is rejected, which stated there would be no statistically significant differences observed in psychometric parameters (as measured via questionnaires regarding quality of life and body image) as a result of diet intervention and/or the presence of the waist circumference metabolic syndrome risk factor.

ANALYSIS OF HYPERTRIGLYCERIDEMIA AS A RISK FACTOR

The hypertriglyceridemia risk factor for metabolic syndrome in women has been defined as a triglyceride level greater than or equal to 1.7 mmol/L. Participants have been identified as either low triglyceride (LTG; <1.7 mmol/L, $N=443$), or high triglyceride (HTG; ≥ 1.7 mmol/L, $N=220$).

Energy Intake

Table 4.23 depicts the time x diet x triglyceride risk factor MANOVA for total energy intake in kcal/kg/day as well as macronutrient intake in g/kg/day. The three-way MANOVA revealed overall time (Wilks' Lambda $p<0.001$), time x diet (Wilks' Lambda $p<0.001$), and time x diet x triglyceride risk factor (Wilks' Lambda $p=0.013$) effects, but no significant time x triglyceride risk factor (Wilks' Lambda $p=0.16$) interaction. Post hoc analysis on the ten-week values was utilized to determine the following: Participants in the HC group and the HTG group consumed significantly more calories relative to body weight (HP 15.9 ± 5.2 , HC 17.4 ± 4.7 kcal/kg/d, $p<0.007$) (LTG 16.2 ± 5.1 , HTG 17.2 ± 4.8 kcal/kg/d, $p=0.042$). Further review of the individual group-combinations revealed that HP-LTG consumed significantly less energy than the other groups (15.2 ± 4.9 kcal/kg/d, $p<0.001$).

Table 4.23: Dietary Intake Before and After Ten Weeks of Exercise and Dietary Intervention Based on the Hypertriglyceridemia Risk Factor Status

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Energy Intake (kcal/kg/day)	HP-LTG	20.2	± 5.7	15.2 ^g	± 4.9				T = 0.001
	HP-HTG	20.9	± 5.8	17.2 ^{cg}	± 5.4				D = 0.042
	HC-LTG	21.2	± 7.0	17.5 ^{cg}	± 5.0				TG = 0.22
	HC-HTG	20.7	± 5.6	17.2 ^g	± 4.1				D x TG = 0.014
	HP	20.4	± 5.8	15.9 ^g	± 5.2	18.4 [†]	± 0.2	T x D = 0.20	
	HC	21.0	± 6.6	17.4 ^{ag}	± 4.7	19.1	± 0.3	T x TG = 0.18	
	LTG	20.6	± 6.3	16.2 ^g	± 5.1	18.5	± 0.2	T x D x TG = 0.35	
	HTG	20.8	± 5.7	17.2 ^{bg}	± 4.8	19.0	± 0.3		
	Time	20.7	± 6.1	16.6*	± 5.0				
Protein Intake (g/kg/day)	HP-LTG	0.83	± 0.2	1.09 ^g	± 0.5				T = 0.001
	HP-HTG	0.85	± 0.2	1.24 ^{cg}	± 0.5				D = 0.001
	HC-LTG	0.84	± 0.2	0.81 ^c	± 0.3				TG = 0.35
	HC-HTG	0.81	± 0.2	0.74 ^d	± 0.2				D x TG = 0.002
	HP	0.83	± 0.2	1.14 ^g	± 0.5	1.00 [†]	± 0.0	T x D = 0.001	
	HC	0.83	± 0.2	0.78 ^a	± 0.3	0.80	± 0.0	T x TG = 0.25	
	LTG	0.83	± 0.2	0.97 ^g	± 0.4	0.89	± 0.0	T x D x TG = 0.030	
	HTG	0.83	± 0.2	1.02 ^g	± 0.5	0.91	± 0.0		
	Time	0.83	± 0.2	0.98*	± 0.4				
Carbohydrate Intake (g/kg/day)	HP-LTG	2.37	± 0.9	1.35 ^g	± 0.6				T = 0.001
	HP-HTG	2.46	± 0.8	1.54 ^{cg}	± 0.8				D = 0.001
	HC-LTG	2.51	± 1.0	2.21 ^{cg}	± 0.8				TG = 0.14
	HC-HTG	2.56	± 0.8	2.19 ^{dg}	± 0.6				D x TG = 0.27
	HP	2.40	± 0.9	1.41 ^g	± 0.7	1.93 [†]	± 0.0	T x D = 0.001	
	HC	2.52	± 0.9	2.20 ^{ag}	± 0.7	2.34	± 0.0	T x TG = 0.87	
	LTG	2.43	± 0.9	1.73 ^g	± 0.8	2.11	± 0.0	T x D x TG = 0.30	
	HTG	2.50	± 0.8	1.82 ^g	± 0.8	2.19	± 0.0		
	Time	2.46	± 0.9	1.76*	± 0.8				
Fat Intake (g/kg/day)	HP-LTG	0.82	± 0.3	0.60 ^g	± 0.3				T = 0.001
	HP-HTG	0.85	± 0.3	0.68 ^{cg}	± 0.3				D = 0.25
	HC-LTG	0.87	± 0.4	0.61 ^g	± 0.2				TG = 0.65
	HC-HTG	0.80	± 0.3	0.60 ^{dfig}	± 0.2				D x TG = 0.008
	HP	0.83	± 0.3	0.63 ^g	± 0.3	0.74	± 0.0	T x D = 0.17	
	HC	0.85	± 0.3	0.60 ^{ag}	± 0.2	0.72	± 0.0	T x TG = 0.039	
	LTG	0.84	± 0.3	0.61 ^g	± 0.2	0.73	± 0.0	T x D x TG = 0.67	
	HTG	0.83	± 0.3	0.65 ^g	± 0.2	0.73	± 0.0		
	Time	0.84	± 0.3	0.62*	± 0.2				
HP = higher protein, HC = higher carbohydrate, LTG = low triglyceride, HTG = high triglyceride risk factor, T = time effect, D = diet effect, TG = triglyceride risk factor effect, D x TG = diet by triglyceride risk factor effect, T x D = time by diet effect, T x TG = time by triglyceride risk factor effect, T x D x TG = time by diet by triglyceride risk factor effect. Values are means ± standard deviations (except group means are means ± standard error) from 247 participants in the HP-LTG group, 124 in the HP-HTG group, 196 in the HC-LTG group, 96 in the HC-HTG group, 371 in the HP total group, 292 in the HC total group, 443 in the LTG group, 220 in the HTG group, and 663 participants total. * Significantly different than baseline, p < 0.05 (univariate). † Significant diet effect, p < 0.05 (univariate). ‡ Significant triglyceride effect, p < 0.05 (univariate). ^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than LTG group, p < 0.05 (post hoc LSD). ^c Significantly different than HP-LTG group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-HTG group, p < 0.05 (post hoc LSD). ^f Significantly different than HC-LTG group, p < 0.05 (post hoc LSD). ^g Significantly different than baseline, p <0.05 (post hoc LSD).									

Participants in the HP group consumed significantly more protein relative to body weight (HP 1.14 ± 0.5 , HC 0.78 ± 0.3 g/kg/d, $p < 0.001$), with no significant differences between the triglyceride risk factor groups. Specifically, the HP-HTG group consumed significantly more protein than all other group-combinations (1.24 ± 0.5 g/kg/d, $p = 0.001$). The HC group consumed significantly more carbohydrate relative to body weight (HP 1.41 ± 0.7 , HC 2.20 ± 0.7 g/kg/d, $p < 0.001$), with no significant difference observed between the risk factor groups. The HC-LTG group consumed significantly more carbohydrate than HP-LTG ($p < 0.001$) and HC-HTG consumed significantly more carbohydrate than HP-HTG ($p < 0.001$), which consumed significantly more carbohydrate than HP-LTG ($p = 0.014$). Finally, the HP group consumed significantly more fat (HP 0.63 ± 0.3 , HC 0.60 ± 0.2 g/kg/d, $p = 0.047$) and the HTG tended to consume a greater amount of fat (LTG 0.61 ± 0.2 , HTG 0.65 ± 0.2 g/kg/d, $p = 0.062$), with the HP-HTG group consuming a significantly greater amount than any other group-combination ($p = 0.015$).

Overall Analysis

Table 4.24 represents the Greenhouse-Geisser univariate and (when applicable) Wilks' Lambda multivariate p-levels for each of the variables and groups measured and analyzed by the various diet and triglyceride risk factor interactions (time, diet, triglyceride risk factor, time x diet, time x triglyceride risk factor, diet x triglyceride risk factor, and time x diet x triglyceride risk factor). The significant findings per variable are discussed below.

Table 4.24: Significance Levels for Triglyceride Risk Factor Analysis per Variable

	Multivariate and Univariate P-Values							Post Hoc Significance							
	T	D	T*D	TG	T*TG	D*TG	T*D*TG	HP-LTG	HP-HTG	HC-LTG	HC-HTG	HP	HC	LTG	HTG
Body Composition	0.001		0.064		0.90		0.34								
Scanned Mass	0.001	0.001	0.007	0.45	0.52	0.07	0.22	g	g	c.g	g	g	a.g	g	g
Fat Mass	0.001	0.001	0.004	0.35	0.58	0.35	0.61	g	g	c.g	g	g	a.g	g	g
Lean Mass	0.001	0.001	0.46	0.74	0.71	0.74	0.12	g	g	c.g	g	g	a.g	g	g
Body Fat	0.001	0.036	0.14	0.27	0.90	0.27	0.74	g	g	g	g	g	g	g	g
Weight	0.001	0.001	0.002	0.42	0.72	0.058	0.14	g	c.g	c.g	g	g	a.g	g	g
Body Mass Index	0.001	0.001	0.002	0.83	0.85	0.29	0.24	g	g	c.g	d.g	g	a.g	g	g
Waist Circumference	0.001	0.001	0.015	0.28	0.79	0.26	0.035	g	g	c.g	g	g	a.g	g	g
Hip Circumference	0.001	0.001	0.48	0.97	0.22	0.34	0.13	g	g	c.g	g	g	a.g	g	g
Resting Energy Expenditure	0.001	0.001	0.324	0.13	0.60	0.53	0.96	g	g	c.g	g	g	a.g	g	g
Hemodynamic	0.001		0.71		0.20		0.66								
Resting Heart Rate	0.001	0.42	0.95	0.035	0.53	0.51	0.67	g	g	g	d.g	g	g	g	g
Systolic Blood Pressure	0.001	0.07	0.46	0.60	0.036	0.35	0.35	g	g		d.g	g	a.g	g	g
Diastolic Blood Pressure	0.001	0.001	0.25	0.65	0.31	0.96	0.27	g		g	d.g	g	a.g	g	g
Blood Lipids	0.001		0.20		0.001		0.07								
Total Cholesterol	0.001	0.61	0.24	0.001	0.45	0.76	0.19	g	c.g	g	f	g	g	b.g	g
LDL Cholesterol	0.001	0.33	0.31	0.001	0.68	0.33	0.08	g	c.g	g	f	g	g	b.g	g
HDL Cholesterol	0.001	0.83	0.60	0.001	0.094	0.81	0.52	g	c.g	g	f	g	g	b.g	g
Triglycerides	0.001	0.28	0.051	0.001	0.001	0.35	0.90		c.g	g	f.g	g	g	b	g
TC/HDL Ratio	0.11	0.78	0.23	0.001	0.16	0.47	0.12	g	c		f			b.g	
Glucose	0.006	0.29	0.50	0.001	0.21	0.63	0.92		c.g		f	g		b	g
Insulin	0.66	0.002	0.67	0.031	0.62	0.98	0.53			c			a		
HOMA	0.98	0.003	0.42	0.010	0.40	0.91	0.22			c			a		
Maximum Strength	0.001		0.53		0.17		0.72								
Bench Press Max Strength	0.001	0.76	0.41	0.69	0.18	0.004	0.96	g	c.g	c.g	g	g	g	g	g
Bench Press Lift Volume	0.89	0.46	0.86	0.84	0.78	0.102	0.17								
Leg Press Max Strength	0.001	0.45	0.51	0.43	0.17	0.045	0.95	g	g	c.g	f.g	g	g	g	g
Leg Press Lift Volume	0.40	0.016	0.15	0.13	0.40	0.23	0.58	g		c			a		
Peak VO ₂	0.001	0.009	0.012	0.85	0.30	0.32	0.67	g	g	c.g	d.g	g	a.g	g	g

Table 4.24: Continued

	Multivariate and Univariate P-Values							Post Hoc Significance							
	T	D	T*D	TG	T*TG	D*TG	T*D* ^a TG	HP- LTG	HP- HTG	HC- LTG	HC- HTG	HP	HC	LTG	HTG
SF36 Questionnaire	0.001		0.076		0.096		0.60								
Physical Function	0.001	0.43	0.67	0.23	0.22	0.24	0.79	g	c	g		g	g	g	
Role Physical	0.52	0.001	0.91	0.08	0.31	0.93	0.60			c	d		a		
Bodily Pain	0.039	0.42	0.13	0.31	0.047	0.47	0.29	g		g		g		g	
General Health	0.001	0.001	0.72	0.66	0.21	0.55	0.18	g		c,g	d,g	g	a,g	g	g
Vital	0.001	0.18	0.13	0.28	0.41	0.14	0.90	g	g	c,g	g	g	g	g	g
Social	0.009	0.001	0.07	0.52	0.66	0.23	0.77	g	g	c		g	a		g
Role Emotional	0.33	0.001	0.11	0.33	0.20	0.85	0.47			c	d		a		
Mental Health	0.001	0.019	0.71	0.58	0.17	0.22	0.18	g	g	g	d,f,g	g	g	g	g
Body Image Questionnaire	0.001		0.72		0.70		0.34								
Appearance Evaluation	0.001	0.55	0.58	0.26	0.71	0.62	0.96	g	g	g	g	g	g	g	g
Appearance Orientation	0.49	0.020	0.36	0.41	0.23	0.96	0.77			c			a		
Body Area Satisfaction Scale	0.001	0.002	0.64	0.74	0.20	0.88	0.033	g	g	c,g	g	g	a,g	g	g
Overweight Preoccupation	0.001	0.29	0.12	0.74	0.98	0.69	0.62	g	g	g	g	g	g	g	g
Self Classified Weight	0.024	0.14	0.43	0.66	0.87	0.39	0.48					g	g	g	
Rosenberg Self Esteem Scale	0.006	0.94	0.38	0.28	0.37	0.20	0.32	g				g			g
Social Physique Anxiety Scale	0.56	0.74	0.83	0.44	0.46	0.85	0.59								

T = time effect, D = diet effect, T x D = time by diet effect, TG = triglyceride risk factor effect, T x TG = time by triglyceride risk factor effect, D x TG = diet by triglyceride risk factor effect, T x D x TG = time by diet by triglyceride risk factor effect. HP = higher protein, HC = higher carbohydrate, LTG = low triglyceride (<1.7 mmol/L), HWC = high triglyceride (≥1.7 mmol/L). N=663 participants unless stated otherwise as follows: REE N=633, Insulin and HOMA N=252, Strength N=596, SF36 N=289, and Body Image N=451. Group breakdowns are as follows unless otherwise stated: 247 in the HP-LTG group, 124 in the HP-HTG group, 196 in the HC-LTG group, 96 in the HC-HTG group, 371 in the HP total group, 292 in the HC total group, 443 in the LTG group, 220 in the HTG group, and 663 participants total; for REE: 239 in the HP-LTG group, 118 in the HP-HTG group, 188 in the HC-LTG group, 88 in the HC-HTG group, 357 in the HP total group, 276 in the HC total group, 427 in the LTG group, 206 in the HTG group, and 633 participants total; for insulin and HOMA: 99 in the HP-LTG group, 50 in the HP-HTG group, 65 in the HC-LTG group, 38 in the HC-HTG group, 149 in the HP total group, 103 in the HC total group, 194 in the LTG group, 88 in the HTG group, and 252 participants total; for strength: 230 in the HP-LTG group, 110 in the HP-HTG group, 177 in the HC-LTG group, 79 in the HC-HTG group, 340 in the HP total group, 256 in the HC total group, 407 in the LTG group, 189 in the HTG group, and 596 participants total; for SF36: 100 in the HP-LTG group, 52 in the HP-HTG group, 84 in the HC-LTG group, 53 in the HC-HTG group, 152 in the HP total group, 137 in the HC total group, 184 in the LTG group, 105 in the HTG group, and 289 participants total; and for Body Image: 177 in the HP-LTG group, 79 in the HP-HTG group, 127 in the HC-LTG group, 68 in the HC-HTG group, 256 in the HP total group, 195 in the HC total group, 304 in the LTG group, 147 in the HTG group, and 451 participants total. Wilks' Lambda Multivariate p-values for the group of variables are listed in bold. Greenhouse-Geisser Univariate p-values are listed for each variable included in the MANOVA (or ANOVA when indicated) analysis. Superscripts indicate significant differences between groups using Post Hoc LSD Pairwise Comparisons. ^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than HTG group, p < 0.05 (post hoc LSD). ^c Significantly different than HP-LTG group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-HTG group, p < 0.05 (post hoc LSD). ^e Significantly different than HC-LTG group, p < 0.05 (post hoc LSD). ^f Significantly different than baseline, p < 0.05 (post hoc LSD).

When a significant three-way interaction was observed, the significant group differences revealed in the post hoc LSD pairwise comparisons analysis are also indicated. Each corresponding hypothesis is also evaluated.

Body Composition

A three-way MANOVA on DEXA variables revealed an overall time effect (Wilks' Lambda $p < 0.001$) and a trend in time x diet (Wilks' Lambda $p = 0.064$), with univariate p-values revealing that participants in the higher protein group experienced a greater loss in both scanned mass (HP -3.9 ± 3.5 , HC -3.0 ± 3.5 kg, $p = 0.007$) and fat mass (HP -3.1 ± 2.7 , HC -2.4 ± 2.8 kg, $p = 0.004$). No significant time x triglyceride risk factor (Wilks' Lambda $p = 0.90$) or time x diet x triglyceride risk factor (Wilks' Lambda $p = 0.34$) interactions were observed. Individual three-way ANOVAs were performed on weight, body mass index, waist circumference, and hip circumference. Significant diet x time interactions were observed indicating a greater decrease in the higher protein group for weight (HP -4.3 ± 3.6 , HC -3.2 ± 3.4 kg, $p = 0.002$), body mass index (HP -1.6 ± 1.3 , HC -1.2 ± 1.3 kg/m², $p = 0.002$), and waist circumference (HP -4.0 ± 5.7 , HC -3.2 ± 5.7 cm, $p = 0.015$). No significant time x triglyceride risk factor interactions were observed for any of these variables. A significant three-way interaction with time x diet x triglyceride risk factor was observed for waist circumference only ($p = 0.035$), with post hoc analysis on the delta change from baseline indicating the HP-HTG group experienced a significantly greater decrease in waist circumference than HC-HTG (HP-HTG -4.6 ± 5.5 , HC-HTG -2.4 ± 4.8 cm, $p = 0.005$). Based on these findings, hypotheses H₁₈ and H₁₉ are accepted, which stated there would

be statistically significant differences observed in body composition as a result of diet intervention, and there would be no statistically significant differences observed in body composition as a result of the presence of the triglyceride metabolic syndrome risk factor, respectively.

Resting Energy Expenditure

The three-way ANOVA for resting energy expenditure and triglyceride risk factor revealed an overall time effect ($p < 0.001$), but no significant interactions. Therefore, hypothesis H_{20} is accepted, which stated there would be no statistically significant differences observed in resting energy expenditure parameters as a result of diet intervention and/or the presence of the triglyceride metabolic syndrome risk factor.

Resting Hemodynamic Parameters

The three-way MANOVA for resting hemodynamic parameters and triglyceride risk factor revealed an overall time effect (Wilks' Lambda $p < 0.001$), but no significant interactions. However, univariate MANOVA on systolic blood pressure revealed a significant time x triglyceride risk factor effect, revealing that the high triglyceride group experienced a significantly greater decrease in systolic blood pressure over the ten-week study (LTG -2.0 ± 14.3 , HTG -5.6 ± 13.8 mmHg, $p = 0.036$). Based on this finding, hypothesis H_{21} is rejected. This hypothesis stated there would be no statistically significant differences observed in resting hemodynamic parameters as a result of diet intervention and/or the presence of the triglyceride metabolic syndrome risk factor.

Blood Lipids

A three-way MANOVA on blood lipids revealed an overall time effect (Wilks' Lambda $p < 0.001$). While the overall time x diet interaction was not significant (Wilks' Lambda $p = 0.20$), analysis of MANOVA univariate tests revealed a significant time x diet effect for triglycerides, with the higher protein group experiencing a significantly greater decrease in triglyceride levels (HP -0.15 ± 0.62 , HC -0.05 ± 0.61 mmol/L, $p = 0.051$). A significant time x triglyceride risk factor interaction (Wilks' Lambda $p < 0.001$) as well as a trend for a time x diet x triglyceride risk factor effect (Wilks' Lambda $p = 0.075$) were also observed. Based on analysis of MANOVA univariate tests, participants identified as having high triglycerides experienced a significantly greater *decrease* in triglyceride levels (LTG $+0.03 \pm 0.4$, HTG -0.37 ± 0.8 mmol/L, $p < 0.001$). Additionally, the LTG group tended to experience a greater decrease in HDL levels than the HTG group (LTG -0.08 ± 0.3 , HTG -0.05 ± 0.2 mmol/L, $p = 0.094$). Finally, post hoc analysis on the time x diet x triglyceride risk factor interaction revealed a trend towards significance for the LDL variable (univariate $p = 0.077$), with the value for the HP-HTG group (3.1 ± 0.8 mmol/L) significantly greater than the HP-LTG group (2.9 ± 0.7 mmol/L, $p = 0.039$), and the HC-HTG group (3.1 ± 0.8 mmol/L) significantly greater than the HC-LTG group (2.9 ± 0.7 , $p < 0.001$) at time point 2. Based on these findings, hypothesis H₂₂ is accepted, which stated there would be statistically significant differences observed in blood lipids as a result of diet intervention and/or the presence of the triglyceride metabolic syndrome risk factor.

Glucose Homeostasis

The three-way ANOVA revealed a time effect for glucose ($p=0.006$), however no significant interactions were observed for glucose, insulin, or HOMA. Therefore hypothesis H_{23} is rejected, which stated there would be statistically significant differences observed in markers of glucose homeostasis as a result of diet intervention and/or the presence of the triglyceride metabolic syndrome risk factor.

Fitness Parameters

The three-way MANOVA on maximum strength revealed an overall time effect (Wilks' Lambda $p<0.001$), but no significant interactions. However, an ANOVA on cardiorespiratory fitness, measured by peak VO_2 , demonstrated a significant time ($p<0.001$) and time x diet effect ($p=0.012$). The HC group experienced a significantly greater increase in peak VO_2 (HP $+1.9\pm3.0$, HC $+2.6\pm3.7$ ml/kg/min, $p=0.012$). Therefore hypothesis H_{24} is rejected, which stated there would be no statistically significant differences observed in fitness parameters as a result of diet intervention and/or the presence of the triglyceride metabolic syndrome risk factor.

Psychometric Analysis

The three-way MANOVA for SF36 demonstrated a time effect (Wilks' Lambda $p<0.001$) and a trend towards significance for both time x diet (Wilks' Lambda $p=0.076$) and time x triglyceride risk factor (Wilks' Lambda $p=0.096$) effect. Review of the MANOVA univariate tests reveals the higher protein group tended to have a greater increase in the social variable (HP $+4.4\pm15.5$, HC $+0.8\pm19.0$, $p=0.073$), and that the LTG group experienced a significantly greater increase in the bodily pain variable (LTG

+4.3±16.9,HTG +0.06±17.4, p=0.047). No significant time x diet x triglyceride risk factor interactions were observed (Wilks' Lambda p=0.60).

For the Body Image analysis, a time effect (Wilks' Lambda p<0.001), but no significant interactions were observed based on the MANOVA multivariate tests. However, review of the MANOVA univariate tests revealed a significant time x diet x triglyceride risk factor interaction for the body area satisfaction scale (p=0.033). Post hoc analysis on the calculated delta changes from baseline revealed the HP-LTG (+0.2±0.4) group experienced significantly less of an increase in body area satisfaction than both HC-LTG (0.3±0.5, p=0.24) and HP-HTG (0.4±0.4, p=0.011). Based on these findings, hypothesis H₂₅ is rejected which stated there would be no statistically significant differences observed in psychometric parameters (as measured via questionnaires regarding quality of life and body image) as a result of diet intervention and/or the presence of the triglyceride metabolic syndrome risk factor.

ANALYSIS OF HDL CHOLESTEROL AS A RISK FACTOR

The HDL cholesterol risk factor for metabolic syndrome in women has been defined as a HDL level less than 1.3 mmol/L. Participants have been identified as either high HDL (HHDL; ≥1.3 mmol/L, N=400), or low HDL (LHDL; <1.3 mmol/L, N=263).

Energy Intake

Table 4.25 depicts the time x diet x HDL risk factor MANOVA for total energy intake in kcals/kg/day as well as macronutrient intake in g/kg/day.

Table 4.25: Dietary Intake Before and After Ten Weeks of Exercise and Dietary Intervention Based on HDL Cholesterol Risk Factor Status

Variable	Group	Baseline			10 Weeks			Group (SEM)			P-level
Energy Intake (kcal/kg/day)	HP-HHDL	20.1	±	5.9	16.1 ^g	±	5.6				T = 0.001
	HP-LHDL	20.8	±	5.6	15.6 ^g	±	4.4				D = 0.002
	HC-HHDL	21.0	±	6.3	17.2 ^{cg}	±	4.7				HDL = 0.57
	HC-LHDL	21.2	±	7.0	17.7 ^{dg}	±	4.7				D x HDL = 0.70
	HP	20.4	±	5.8	15.9 ^g	±	5.2	18.2 [†]	±	0.2	T x D = 0.055
	HC	21.0	±	6.6	17.4 ^{ag}	±	4.7	19.3	±	0.3	T x HDL = 0.39
	HHDL	20.5	±	6.1	16.6 ^g	±	5.3	18.6	±	0.2	T x D x HDL = 0.16
	LHDL	21.0	±	6.2	16.5 ^g	±	4.6	18.8	±	0.3	
Time	20.7	±	6.1	16.6 [*]	±	5.0					
Protein Intake (g/kg/day)	HP-HHDL	0.82	±	0.2	1.17 ^g	±	0.6				T = 0.001
	HP-LHDL	0.85	±	0.2	1.10 ^g	±	0.4				D = 0.001
	HC-HHDL	0.84	±	0.2	0.79 ^c	±	0.3				HDL = 0.18
	HC-LHDL	0.80	±	0.2	0.78 ^d	±	0.2				D x HDL = 0.93
	HP	0.83	±	0.2	1.14 ^g	±	0.5	0.99 [†]	±	0.0	T x D = 0.001
	HC	0.83	±	0.2	0.78 ^a	±	0.3	0.80	±	0.0	T x HDL = 0.25
	HHDL	0.83	±	0.2	1.00 ^g	±	0.5	0.91	±	0.0	T x D x HDL = 0.053
	LHDL	0.83	±	0.2	0.96 ^g	±	0.4	0.88	±	0.0	
Time	0.83	±	0.2	0.98 [*]	±	0.4					
Carbohydrate Intake (g/kg/day)	HP-HHDL	2.37	±	0.9	1.38 ^g	±	0.7				T = 0.001
	HP-LHDL	2.45	±	0.8	1.46 ^g	±	0.6				D = 0.001
	HC-HHDL	2.48	±	0.9	2.19 ^{cg}	±	0.7				HDL = 0.12
	HC-LHDL	2.60	±	1.0	2.23 ^{dg}	±	0.7				D x HDL = 1.0
	HP	2.40	±	0.9	1.41 ^g	±	0.7	1.91 [†]	±	0.0	T x D = 0.001
	HC	2.52	±	0.9	2.20 ^{ag}	±	0.7	2.37	±	0.0	T x HDL = 0.61
	HHDL	2.42	±	0.9	1.74 ^g	±	0.8	2.10	±	0.0	T x D x HDL = 0.69
	LHDL	2.51	±	0.9	1.79 ^g	±	0.7	2.18	±	0.0	
Time	2.46	±	0.9	1.76 [*]	±	0.8					
Fat Intake (g/kg/day)	HP-HHDL	0.82	±	0.3	0.65 ^g	±	0.3				T = 0.001
	HP-LHDL	0.85	±	0.3	0.60 ^{cg}	±	0.2				D = 0.95
	HC-HHDL	0.85	±	0.3	0.59 ^{cg}	±	0.2				HDL = 0.98
	HC-LHDL	0.84	±	0.4	0.63 ^g	±	0.2				D x HDL = 0.33
	HP	0.83	±	0.3	0.63 ^g	±	0.3	0.73	±	0.0	T x D = 0.27
	HC	0.85	±	0.3	0.60 ^g	±	0.2	0.73	±	0.0	T x HDL = 0.57
	HHDL	0.83	±	0.3	0.62 ^g	±	0.3	0.73	±	0.0	T x D x HDL = 0.013
	LHDL	0.84	±	0.3	0.61 ^g	±	0.2	0.73	±	0.0	
Time	0.84	±	0.3	0.62 [*]	±	0.2					
HP = higher protein, HC = higher carbohydrate, HHDL = high HDL cholesterol, LHDL = low HDL cholesterol risk factor, T = time effect, D = diet effect, HDL = HDL cholesterol risk factor effect, D x HDL = diet by HDL cholesterol risk factor effect, T x D = time by diet effect, T x HDL = time by HDL cholesterol risk factor effect, T x D x HDL = time by diet by HDL cholesterol risk factor effect. Values are means ± standard deviations (except group means are means ± standard error) from 220 participants in the HP-HHDL group, 151 in the HP-LHDL group, 180 in the HC-HHDL group, 112 in the HC-LHDL group, 371 in the HP total group, 292 in the HC total group, 400 in the HHDL group, 263 in the LHDL group, and 663 participants total. * Significantly different than baseline, p < 0.05 (univariate). † Significant diet effect, p < 0.05 (univariate). ‡ Significant HDL cholesterol effect, p < 0.05 (univariate). ^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than HHDL group, p < 0.05 (post hoc LSD). ^c Significantly different than HP-HHDL group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-LHDL group, p < 0.05 (post hoc LSD). ^e Significantly different than HC-HHDL group, p < 0.05 (post hoc LSD). ^f Significantly different than baseline, p < 0.05 (post hoc LSD).											

The three-way MANOVA revealed an overall time (Wilks' Lambda $p<0.001$), time x diet (Wilks' Lambda $p<0.001$), and time x diet x HDL risk factor (Wilks' Lambda $p=0.020$) effect, but no significant time x HDL risk factor (Wilks' Lambda $p=0.68$) interaction. Post hoc analysis on the ten-week values was utilized to determine the following: Participants in the HC group consumed significantly more calories (HP 15.9 ± 5.2 , HC 17.4 ± 4.7 kcal/kg/d, $p<0.001$) and carbohydrate (HP 1.41 ± 0.7 , HC 2.20 ± 0.7 g/kg/d, $p<0.001$), while the HP group consumed significantly more protein (HP 1.14 ± 0.5 , HC 0.78 ± 0.3 g/kg/d, $p<0.001$) relative to body weight. Regarding specific group-combinations, energy intake was significantly greater for HC-HHDL compared to HP-HHDL ($p=0.029$), and HC-LHDL was significantly greater than HP-LHDL ($p=0.001$). For protein intake, HP-HHDL was significantly greater than HC-HHDL ($p<0.001$), and HP-LHDL was significantly greater than HC-LHDL ($p<0.001$). For carbohydrate intake, HC-HHDL was significantly greater than HP-HHDL ($p<0.001$), and HC-LHDL was significantly greater than HP-LHDL ($p<0.001$). Regarding fat intake, while there were no significant differences between the diet or HDL groups, the HP-HHDL group consumed significantly more fat than both HP-LHDL ($p=0.021$) and HC-HHDL ($p=0.006$).

Overall Analysis

Table 4.26 represents the Greenhouse-Geisser univariate and (when applicable) Wilks' Lambda multivariate p-levels for each of the variables and groups measured and analyzed by the various diet and HDL risk factor interactions (time, diet, HDL risk factor, time x diet, time x HDL risk factor, diet x HDL risk factor, and time x diet x HDL

risk factor). The significant findings per variable are discussed below. When a significant three-way interaction was observed, the significant group differences revealed in the post hoc LSD pairwise comparisons analysis are also indicated. Each corresponding hypothesis is also evaluated.

Body Composition

The three-way MANOVA on DEXA variables revealed a significant time (Wilks' Lambda $p < 0.001$) and time x diet effect (Wilks' Lambda $p = 0.033$), but no significant time x HDL risk factor (Wilks' Lambda $p = 0.68$) or time x diet x HDL risk factor (Wilks' Lambda $p = 0.42$) interactions. Review of the MANOVA univariate tests revealed the higher protein group resulted in a significantly greater decrease in scanned mass (HP -3.9 ± 3.5 , HC -3.0 ± 3.5 kg, $p = 0.002$) and fat mass (HP -3.1 ± 2.7 , HC -2.4 ± 2.8 kg, $p = 0.003$).

Individual three-way ANOVAs were used for weight, body mass index, and waist and hip circumference. Significant univariate time x diet effects were observed for weight, body mass index, and a trend for waist circumference. The higher protein group experienced a significantly greater decrease in weight (HP -4.3 ± 3.6 , HC -3.2 ± 3.4 kg, $p = 0.001$), body mass index (HP -1.6 ± 1.3 , HC -1.2 ± 1.3 kg/m², $p = 0.001$), and tended to experience a greater decrease in waist circumference (HP -4.0 ± 5.7 , HC -3.2 ± 5.7 cm, $p = 0.068$).

Table 4.26: Significance Levels for HDL Cholesterol Risk Factor Analysis per Variable

	Multivariate and Univariate P-Values							Post Hoc Significance							
	T	D	T*D	HDL	T*HDL	D*HDL	T*D*HDL	HP-HHDL	HP-LHDL	HC-HHDL	HC-LHDL	HP	HC	HHDL	LHDL
Body Composition	0.001		0.033		0.68		0.42								
Scanned Mass	0.001	0.001	0.002	0.14	0.35	0.37	0.84		g	c	d	g	a,g	g	g
Fat Mass	0.001	0.001	0.003	0.93	0.35	0.29	0.47		g	c		g	a,g	b,g	g
Lean Mass	0.001	0.001	0.15	0.001	0.80	0.61	0.45		c,g	c	d,f	g	a,g	b,g	g
Body Fat	0.001	0.06	0.21	0.003	0.48	0.33	0.46		c,g			g	g	g	g
Weight	0.001	0.001	0.001	0.15	0.40	0.38	0.77	g	g	c,g	d,g	g	a,g	g	g
Body Mass Index	0.001	0.001	0.001	0.16	0.43	0.19	0.67	g	g	c,g	d,g	g	a,g	g	g
Waist Circumference	0.001	0.001	0.07	0.055	0.98	0.24	0.87	g	g	c,g	d,f,g	g	a,g	g	g
Hip Circumference	0.001	0.001	0.15	0.88	0.97	0.38	0.42	g	g	c,g	g	g	a,g	g	g
Resting Energy Expenditure	0.001	0.001	0.26	0.003	0.36	0.53	0.80	g	c,g	c,g	d	g	a,g	b,g	g
Hemodynamic	0.001		0.81		0.053		0.15								
Resting Heart Rate	0.001	0.23	0.88	0.06	0.07	0.47	0.92	g	g	g	g	g	g	g	g
Systolic Blood Pressure	0.001	0.09	0.39	0.12	0.06	0.58	0.06	g	g		d,f,g	g	a,g	b,g	g
Diastolic Blood Pressure	0.001	0.001	0.43	0.12	0.06	0.35	0.77		g	c,g	d,f,g	g	a,g	b,g	g
Blood Lipids	0.001		0.34		0.001		0.48								
Total Cholesterol	0.001	0.68	0.37	0.001	0.001	0.53	0.96	g	c	g		g	g	b,g	
LDL Cholesterol	0.001	0.42	0.67	0.77	0.001	0.39	0.66	g		g		g	g	g	
HDL Cholesterol	0.001	0.96	0.60	0.001	0.001	0.54	0.88	g	c	g	f	g	g	b,g	g
Triglycerides	0.001	0.52	0.047	0.001	0.09	0.72	0.94	g	c,g		f	g		b,g	g
TC/HDL Ratio	0.18	0.66	0.42	0.001	0.001	0.59	0.40	g	c,g	g	f			b,g	
Glucose	0.032	0.23	0.50	0.18	0.16	0.14	0.39	g	c			g		b,g	
Insulin	0.79	0.001	0.52	0.25	0.13	0.93	0.06			c			a		
HOMA	0.93	0.002	0.37	0.12	0.14	0.97	0.030			c	g		a		
Maximum Strength	0.001		0.44		0.41		0.30								
Bench Press Max Strength	0.001	0.39	0.25	0.001	0.38	0.031	0.20	g	g	c,g	f,g	g	g	b,g	g
Bench Press Lift Volume	0.94	0.48	0.68	0.001	0.44	0.031	0.17				f			b	
Leg Press Max Strength	0.001	0.31	0.61	0.001	0.17	0.016	0.36	g	g	c,g	f,g	g	g	b,g	g
Leg Press Lift Volume	0.13	0.016	0.14	0.14	0.16	0.004	0.28			c	f	g	a		
Peak VO ₂	0.002	0.015	0.012	0.30	0.024	0.80	0.98	g	g	c,g	d,g	g	a,g	g	g

Table 4.26: Continued

	Multivariate and Univariate P-Values							Post Hoc Significance							
	T	D	T*D	HDL	T*HDL	D*HDL	T*D*HDL	HP-HHDL	HP-LHDL	HC-HHDL	HC-LHDL	HP	HC	HHDL	LHDL
SF36 Questionnaire	0.001		0.13		0.38		0.80								
Physical Function	0.001	0.61	0.61	0.75	0.14	0.64	0.38	g	g		g	g	g	g	g
Role Physical	0.42	0.001	0.92	0.06	0.36	0.53	0.30			c	d		a	b	
Bodily Pain	0.004	0.35	0.23	0.06	0.11	0.26	0.58		g		f	g		b	g
General Health	0.001	0.001	0.47	0.11	0.66	0.37	0.64	g	g	c,g	d,g	g	a,g	g	g
Vital	0.001	0.12	0.11	0.48	0.40	0.18	0.75	g	g	c,g	g	g	g	g	g
Social	0.024	0.001	0.09	0.018	0.11	0.39	0.69	g		c	f	g	a	b,g	
Role Emotional	0.45	0.001	0.12	0.22	0.73	1.0	0.31			c	d		a		
Mental Health	0.001	0.035	0.67	0.65	0.75	0.93	0.17	g	g	g	g	g	g	g	g
Body Image Questionnaire	0.001		0.51		0.72		0.84								
Appearance Evaluation	0.001	0.58	0.46	1.0	0.78	0.30	0.42	g	g	g	g	g	g	g	g
Appearance Orientation	0.20	0.010	0.35	0.83	0.59	0.48	0.52				d		a		
Body Area Satisfaction Scale	0.001	0.001	0.20	0.55	0.75	0.62	0.94	g	g	c,g	d,g	g	a,g	g	g
Overweight Preoccupation	0.001	0.30	0.09	0.91	0.29	0.76	0.18	g	g	g	g	g	g	g	g
Self Classified Weight	0.007	0.105	0.57	0.53	0.21	0.33	0.92			c		g			g
Rosenberg Self Esteem Scale	0.008	0.55	0.29	0.16	0.81	0.77	0.42	g				g		g	
Social Physique Anxiety Scale	0.58	0.80	0.91	0.19	0.40	0.025	0.90				f				
<p>T = time effect, D = diet effect, T x D = time by diet effect, HDL = HDL cholesterol risk factor effect, T x HDL = time by HDL cholesterol risk factor effect, D x HDL = diet by HDL cholesterol risk factor effect, T x D x HDL = time by diet by HDL cholesterol risk factor effect. HP = higher protein, HC = higher carbohydrate, HHDL = high HDL cholesterol (≥ 1.3 mmol/L), LHDL = low HDL cholesterol (< 1.3 mmol/L). N=663 participants unless stated otherwise as follows: REE N=633, Insulin and HOMA N=252, Strength N=596, SF36 N=289, and Body Image N=451.</p> <p>Group breakdowns are as follows unless otherwise stated: 220 in the HP-HHDL group, 151 in the HP-LHDL group, 180 in the HC-HHDL group, 112 in the HC-LHDL group, 371 in the HP total group, 292 in the HC total group, 400 in the HHDL group, 263 in the LHDL group, and 663 participants total; for REE: 210 in the HP-HHDL group, 147 in the HP-LHDL group, 170 in the HC-HHDL group, 106 in the HC-LHDL group, 357 in the HP total group, 276 in the HC total group, 380 in the HHDL group, 253 in the LHDL group, and 633 participants total; for insulin and HOMA: 83 in the HP-HHDL group, 66 in the HP-LHDL group, 62 in the HC-HHDL group, 41 in the HC-LHDL group, 149 in the HP total group, 103 in the HC total group, 145 in the HHDL group, 107 in the LHDL group, and 252 participants total; for strength: 201 in the HP-HHDL group, 139 in the HP-LHDL group, 158 in the HC-HHDL group, 98 in the HC-LHDL group, 340 in the HP total group, 256 in the HC total group, 359 in the HHDL group, 237 in the LHDL group, and 596 participants total; for SF36: 89 in the HP-HHDL group, 63 in the HP-LHDL group, 83 in the HC-HHDL group, 54 in the HC-LHDL group, 152 in the HP total group, 137 in the HC total group, 172 in the HHDL group, 117 in the LHDL group, and 289 participants total; for Body Image: 150 in the HP-HHDL group, 106 in the HP-LHDL group, 120 in the HC-HHDL group, 75 in the HC-LHDL group, 256 in the HP total group, 195 in the HC total group, 270 in the HHDL group, 181 in the LHDL group, and 451 participants total.</p> <p>Wilks' Lambda Multivariate p-values for the group of variables are listed in bold. Greenhouse-Geisser Univariate p-values are listed for each variable included in the MANOVA (or ANOVA when indicated) analysis. Superscripts indicate significant differences between groups using Post Hoc LSD Pairwise Comparisons. ^a Significantly different than HP group, $p < 0.05$ (post hoc LSD). ^b Significantly different than LHDL group, $p < 0.05$ (post hoc LSD). ^c Significantly different than HP-HHDL group, $p < 0.05$ (post hoc LSD). ^d Significantly different than HP-LHDL group, $p < 0.05$ (post hoc LSD). ^f Significantly different than HC-HHDL group, $p < 0.05$ (post hoc LSD). ^g Significantly different than baseline, $p < 0.05$ (post hoc LSD).</p>															

No significant time x HDL risk factor or time x diet x HDL risk factor interactions were observed for any of these variables. Based on these findings, hypotheses H₂₆ and H₂₇ are both accepted, which stated there would be statistically significant differences observed in body composition as a result of diet intervention, and there would be no statistically significant differences observed in body composition as a result of the presence of the HDL metabolic syndrome risk factor, respectively.

Resting Energy Expenditure

The three-way ANOVA for resting energy expenditure and HDL risk factor revealed an overall time effect ($p < 0.001$), but no significant interactions. Therefore, hypothesis H₂₈ is accepted, which stated there would be no statistically significant differences observed in resting energy expenditure parameters as a result of diet intervention and/or the presence of the HDL metabolic syndrome risk factor.

Resting Hemodynamic Parameters

The three-way MANOVA on the resting hemodynamic parameters revealed a significant time (Wilks' Lambda $p < 0.001$) and time x HDL risk factor effect (Wilks' Lambda $p = 0.053$), but no significant time x diet (Wilks' Lambda $p = 0.81$) or time x diet x HDL risk factor (Wilks' Lambda $p = 0.15$) interactions. Based on MANOVA univariate tests, there was a tendency for a greater decrease in the LHDL group for resting heart rate (HHDL -2.4 ± 10.2 , LHDL -3.9 ± 10.5 bpm, $p = 0.070$), systolic blood pressure (HHDL -2.1 ± 14.9 , LHDL -4.0 ± 14.5 mmHg, $p = 0.061$), and diastolic blood pressure (HHDL -1.4 ± 9.8 , LHDL -2.8 ± 9.8 mmHg, $p = 0.063$). The univariate tests also revealed a trend towards significance for time x diet x HDL risk factor in systolic blood

pressure ($p=0.06$). Post hoc analysis of the calculated delta change from baseline revealed the HC-LHDL group experienced a significantly greater decrease in systolic blood pressure than HC-HHDL (HC-HHDL -1.4 ± 14.8 , HC-LHDL -5.8 ± 14.8 mmHg, $p=0.013$). Based on these findings, hypothesis H_{29} is rejected, which stated there would be no statistically significant differences observed in resting hemodynamic parameters as a result of diet intervention and/or the presence of the HDL metabolic syndrome risk factor.

Blood Lipids

The three-way MANOVA on blood lipids revealed a significant time (Wilks' Lambda $p<0.001$) and time x HDL risk factor (Wilks' Lambda $p<0.001$), but no significant time x diet (Wilks' Lambda $p=0.34$) or time x diet x HDL risk factor (Wilks' Lambda $p=0.48$) interactions. However, upon reviewing the MANOVA univariate tests, a significant time x diet effect was observed for triglycerides, with the higher protein group experiencing a significantly greater decrease (HP -0.15 ± 0.62 , HC -0.05 ± 0.60 mmol/L, $p=0.047$). Participants with high HDL cholesterol experienced significantly greater reductions in total cholesterol (HHDL -0.33 ± 0.8 , LHDL -0.02 ± 0.7 mmol/L, $p<0.001$) and LDL cholesterol (HHDL -0.22 ± 0.6 , LHDL -0.03 ± 0.6 mmol/L, $p<0.001$), and a significantly greater increase in total cholesterol / HDL ratio (HHDL $+0.14\pm0.6$, LHDL -0.08 ± 0.7 , $p<0.001$). Participants in the low HDL group experienced a significantly greater increase in HDL cholesterol (HHDL -0.14 ± 0.2 , LHDL $+0.04\pm0.19$ mmol/L, $p<0.001$) and tended to have a greater decrease in triglycerides (HHDL -0.07 ± 0.5 , LHDL -0.15 ± 0.7 mmol/L, $p=0.092$). Based on these findings hypothesis H_{30}

is accepted, which stated there would be statistically significant differences observed in blood lipids as a result of diet intervention and/or the presence of the HDL metabolic syndrome risk factor.

Glucose Homeostasis

The three-way ANOVA for glucose revealed a time effect ($p=0.032$), but no significant interaction effects. Likewise, time x diet and time x HDL risk factor interactions were not significant for insulin and HOMA. However, the time x diet x HDL risk factor interaction revealed a trend towards significance for insulin ($p=0.057$), and significance for HOMA ($p=0.030$). Post hoc analysis on the calculated delta changes from baseline revealed a significant *decrease* in insulin for the HC-HHDL group compared to an *increase* for HC-LHDL (HC-HHDL $+1.8 \pm 12.0$, HC-LHDL -2.3 ± 16.0 uIU/mL, $p=0.028$). It also revealed the HC-LHDL group experienced a significant decrease in HOMA (-1.0 ± 6.7) compared to significant increases for both HP-LHDL ($+0.3 \pm 1.9$, $p=0.044$) and HC-HHDL ($+0.6 \pm 3.6$, $p=0.019$). Based on these findings, hypothesis H_{31} which stated there would be statistically significant differences observed in markers of glucose homeostasis as a result of diet intervention is accepted, and hypothesis H_{32} , which stated there would be no statistically significant differences observed in markers of glucose homeostasis as a result of the presence of the HDL metabolic syndrome risk factor, was rejected.

Fitness Parameters

The three-way MANOVA on muscular fitness variables revealed an overall time effect (Wilks' Lambda $p<0.001$) but no significant interactions. The three-way ANOVA

on cardiorespiratory fitness, measured by peak VO_2 , demonstrated a significant time ($p<0.001$), time x diet ($p=0.012$), and time x HDL risk factor ($p=0.024$) effect, but no significant time x diet x HDL risk factor effect (Greenhouse-Geisser $p=0.98$). The HC and the HHDL groups both experienced a significantly greater increase in peak VO_2 (HP $+1.9\pm3.0$, HC $+2.6\pm3.7$ ml/kg/min, $p=0.012$), (HHDL $+2.6\pm3.4$, LHDL $\pm2.2\pm3.3$ ml/kg/min, $p=0.024$). However, these individual group findings did not prove significant when combined into a three-way interaction. Based on the findings from the cardiorespiratory fitness analysis, hypothesis H_{33} is rejected. This hypothesis stated that there would be no statistically significant differences observed in fitness parameters as a result of diet intervention and/or the presence of the HDL metabolic syndrome risk factor.

Psychometric Analysis

The three-way MANOVA analyses on the SF36 – Quality of Life and the Body Image Questionnaires both revealed a significant time effect (Wilks' Lambda $p<0.001$), but no significant overall interactions. However, review of the MANOVA univariate tests revealed a trend towards significance for the time x diet interaction for the social variable within the SF36, and the overweight preoccupation variable within the Body Image Questionnaire. The higher protein group tended to have a greater increase in the social aspect (HP $+4.4\pm15.5$, HC $+0.8\pm19.0$, $p=0.09$) as well as in overweight preoccupation (HP $+0.5\pm0.7$, HC $+0.4\pm0.7$, $p=0.09$). Since these values were only a trend towards significance, hypothesis H_{34} is still accepted. This hypothesis stated there would be no statistically significant differences observed in psychometric parameters (as

measured via questionnaires regarding quality of life and body image) as a result of diet intervention and/or the presence of the HDL metabolic syndrome risk factor.

ANALYSIS OF HYPERTENSION AS A RISK FACTOR

The hypertension risk factor for metabolic syndrome in women has been defined as a blood pressure value greater than or equal to 135 mmHg systolic and/or 85 mmHg diastolic. Participants have been identified as either low blood pressure (LBP; <135/85 mm/Hg, N=419), or high blood pressure (HBP; \geq 135/85 mm/, N=244).

Energy Intake

Table 4.27 depicts the time x diet x blood pressure risk factor MANOVA for total energy intake in kcals/kg/day as well as macronutrient intake in g/kg/day. The three-way MANOVA revealed an overall time (Wilks' Lambda $p < 0.001$) and time x diet (Wilks' Lambda $p < 0.001$) effect, but no significant time x blood pressure risk factor (Wilks' Lambda $p = 0.87$) or time x diet x blood pressure risk factor (Wilks' Lambda $p = 0.84$) interactions. Post hoc analysis on the ten-week values was utilized to determine the following: Participants in the HC group consumed significantly more calories (HP 15.9 ± 5.2 , HC 17.4 ± 4.7 kcal/kg/d, $p = 0.014$) and carbohydrate (HP 1.41 ± 0.7 , HC 2.20 ± 0.7 g/kg/d, $p < 0.001$), while the HP group consumed significantly more protein (HP 1.14 ± 0.5 , HC 0.78 ± 0.3 g/kg/d, $p < 0.001$) relative to body weight. Regarding specific group-combinations, energy intake was significantly greater for HC-LBP compared to HP-LBP ($p = 0.008$), and HC-HBP was significantly greater than HP-HBP ($p = 0.009$). For protein intake, HP-LBP was significantly greater than HC-LBP ($p < 0.001$), and HP-HBP was significantly greater than HC-HBP ($p < 0.001$).

Table 4.27: Dietary Intake Before and After Ten Weeks of Exercise and Dietary Intervention Based on the Hypertension Risk Factor Status

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Energy Intake (kcal/kg/day)	HP-LBP	20.86	± 5.66	16.27 ^g	± 5.30				T = 0.001
	HP-HBP	19.73	± 5.87	15.33 ^g	± 4.90				D = 0.003
	HC-LBP	21.09	± 6.59	17.57 ^{c,g}	± 4.88				BP = 0.06
	HC-HBP	20.94	± 6.58	17.03 ^{d,g}	± 4.36				D x BP = 0.35
	HP	20.41	± 5.76	15.90 ^g	± 5.16	18.05 [†]	± 0.24		T x D = 0.15
	HC	21.04	± 6.58	17.39 ^{a,g}	± 4.71	19.16	± 0.28		T x BP = 0.85
	LBP	20.97	± 6.11	16.88 ^g	± 5.14	18.95	± 0.22		T x D x BP = 0.60
	HBP	20.21	± 6.17	16.00 ^g	± 4.76	18.26 [‡]	± 0.29		
	Time	20.69	± 6.14	16.56 [*]	± 5.02				
Protein Intake (g/kg/day)	HP-LBP	0.86	± 0.24	1.17 ^g	± 0.52				T = 0.001
	HP-HBP	0.80 ^c	± 0.22	1.10 ^g	± 0.43				D = 0.001
	HC-LBP	0.83	± 0.23	0.80 ^c	± 0.28				BP = 0.027
	HC-HBP	0.82	± 0.22	0.75 ^d	± 0.21				D x BP = 0.41
	HP	0.83	± 0.23	1.14 ^g	± 0.49	0.98 [†]	± 0.01		T x D = 0.001
	HC	0.83	± 0.23	0.78 ^a	± 0.26	0.80	± 0.02		T x BP = 0.44
	LBP	0.84	± 0.24	1.00 ^g	± 0.46	0.91	± 0.01		T x D x BP = 0.82
	HBP	0.81	± 0.22	0.96 ^g	± 0.40	0.87 [‡]	± 0.02		
	Time	0.83	± 0.23	0.98 [*]	± 0.44				
Carbohydrate Intake (g/kg/day)	HP-LBP	2.45	± 0.85	1.44 ^g	± 0.68				T = 0.001
	HP-HBP	2.32	± 0.88	1.37 ^g	± 0.62				D = 0.001
	HC-LBP	2.53	± 0.92	2.23 ^{c,g}	± 0.73				BP = 0.15
	HC-HBP	2.51	± 0.97	2.15 ^{d,g}	± 0.66				D x BP = 0.57
	HP	2.40	± 0.87	1.41 ^g	± 0.66	1.90 [†]	± 0.03		T x D = 0.001
	HC	2.52	± 0.93	2.20 ^{a,g}	± 0.71	2.36	± 0.04		T x BP = 0.97
	LBP	2.49	± 0.88	1.81	± 0.81	2.16	± 0.03		T x D x BP = 0.45
	HBP	2.40	± 0.92	1.68	± 0.74	2.09	± 0.04		
	Time	2.46	± 0.90	1.76 [*]	± 0.79				
Fat Intake (g/kg/day)	HP-LBP	0.85	± 0.28	0.65 ^g	± 0.28				T = 0.001
	HP-HBP	0.81	± 0.27	0.61 ^g	± 0.23				D = 0.95
	HC-LBP	0.85	± 0.33	0.61 ^g	± 0.22				BP = 0.19
	HC-HBP	0.85	± 0.33	0.60 ^g	± 0.20				D x BP = 0.30
	HP	0.83	± 0.27	0.63 ^g	± 0.26	0.73	± 0.01		T x D = 0.11
	HC	0.85	± 0.33	0.60 ^g	± 0.21	0.73	± 0.01		T x BP = 0.96
	LBP	0.85	± 0.30	0.63 ^g	± 0.25	0.74	± 0.01		T x D x BP = 0.95
	HBP	0.82	± 0.29	0.60 ^g	± 0.22	0.71	± 0.01		
	Time	0.84	± 0.30	0.62 [*]	± 0.24				

HP = higher protein, HC = higher carbohydrate, LBP = low blood pressure, HBP = high blood pressure risk factor, T = time effect, D = diet effect, BP = blood pressure risk factor effect, D x BP = diet by blood pressure risk factor effect, T x D = time by diet effect, T x BP = time by blood pressure risk factor effect, T x D x BP = time by diet by blood pressure risk factor effect.

Values are means ± standard deviations (except group means are means ± standard error) from 233 participants in the HP-LBP group, 148 in the HP-HBP group, 196 in the HC-LBP group, 96 in the HC-HBP group, 371 in the HP total group, 292 in the HC total group, 419 in the LBP group, 244 in the HBP group, and 663 participants total.

* Significantly different than baseline, $p < 0.05$ (univariate). † Significant diet effect, $p < 0.05$ (univariate). ‡ Significant blood pressure effect, $p < 0.05$ (univariate). ^a Significantly different than HP group, $p < 0.05$ (post hoc LSD). ^b Significantly different than LBP group, $p < 0.05$ (post hoc LSD). ^c Significantly different than HP-LBP group, $p < 0.05$ (post hoc LSD). ^d Significantly different than HP-HBP group, $p < 0.05$ (post hoc LSD). ^e Significantly different than HC-LBP group, $p < 0.05$ (post hoc LSD). ^g Significantly different than baseline, $p < 0.05$ (post hoc LSD).

For carbohydrate intake, HC-LBP was significantly greater than HP-LBP ($p<0.001$), and HC-HBP was significantly greater than HP-HBP ($p<0.001$). No significant differences were observed between group-combinations regarding fat intake.

Overall Analysis

Table 4.28 represents the Greenhouse-Geisser univariate and (when applicable) Wilks' Lambda multivariate p-levels for each of the variables and groups measured and analyzed by the various diet and blood pressure risk factor interactions (time, diet, blood pressure risk factor, time x diet, time x blood pressure risk factor, diet x blood pressure risk factor, and time x diet x blood pressure risk factor). The significant findings per variable are discussed below. When a significant three-way interaction was observed, the significant group differences revealed in the post hoc LSD pairwise comparisons analysis are also indicated. Each corresponding hypothesis is also evaluated.

Body Composition

The three-way MANOVA on DEXA variables revealed an overall time (Wilks' Lambda $p<0.001$) and time x diet (Wilks' Lambda $p=0.045$) effect, but no significant time x blood pressure risk factor (Wilks' Lambda $p=0.52$) or time x diet x blood pressure risk factor (Wilks' Lambda $p=0.32$) interactions. Review of the MANOVA univariate tests for DEXA variables demonstrated that for time x diet, the higher protein group experienced a significantly greater decrease in scanned mass (HP -3.9 ± 3.5 , HC -3.0 ± 3.5 kg, $p=0.002$) and fat mass (HP -3.1 ± 2.7 , HC -2.4 ± 2.8 kg, $p=0.007$). Additionally, regarding time x diet x blood pressure risk factor, body fat was significant at $p=0.045$.

Table 4.28: Significance Levels for Blood Pressure Risk Factor Analysis per Variable

	Multivariate and Univariate P-Values							Post Hoc Significance							
	T	D	T*D	BP	T*BP	D*BP	T*D*BP	HP-LBP	HP-HBP	HC-LBP	HC-HBP	HP	HC	LBP	HBP
Body Composition	0.001		0.045		0.52		0.32								
Scanned Mass	0.001	0.001	0.002	0.014	0.17	0.47	0.94	g	c,g	c,g	d,g	g	a,g	b,g	g
Fat Mass	0.001	0.001	0.007	0.018	0.13	0.75	0.32	g	c,g	c,g	d,g	g	a,g	b,g	g
Lean Mass	0.001	0.001	0.110	0.029	0.78	0.26	0.22	g	c,g	c,g	d,g	g	a,g	b,g	g
Body Fat	0.001	0.11	0.41	0.15	0.52	0.42	0.045	g	g	g	g	g	g	g	g
Weight	0.001	0.001	0.001	0.016	0.07	0.46	0.63	g	c,g	c,g	d,g	g	a,g	b,g	g
Body Mass Index	0.001	0.001	0.001	0.013	0.09	0.38	0.59	g	c,g	c,g	d,g	g	a,g	b,g	g
Waist Circumference	0.001	0.001	0.10	0.001	0.48	0.45	0.89	g	c,g	c,g	d,g	g	a,g	b,g	g
Hip Circumference	0.001	0.001	0.51	0.005	0.29	0.89	0.07	g	c,g	c,g	d,g	g	a,g	b,g	g
Resting Energy Expenditure	0.001	0.001	0.20	0.005	0.67	0.51	0.29	g	c,g	c,g	d	g	a,g	b,g	g
Hemodynamic	0.001		0.48		0.001		0.86								
Resting Heart Rate	0.001	0.63	0.87	0.11	0.61	0.08	0.93	g	g	g	g	g	g	g	g
Systolic Blood Pressure	0.001	0.54	0.22	0.001	0.001	0.88	0.68		c,g		f,g	g	g	b	g
Diastolic Blood Pressure	0.001	0.004	0.17	0.001	0.001	0.52	0.59	g	c,g	c	f,g	g	a,g	b	g
Blood Lipids	0.001		0.40		0.028		0.96								
Total Cholesterol	0.001	0.38	0.53	0.003	0.09	0.28	0.99	g	g	g	f,g	g	g	b,g	g
LDL Cholesterol	0.001	0.32	0.66	0.015	0.38	0.44	0.76	g	g	g	g	g	g	g	g
HDL Cholesterol	0.001	0.61	0.85	0.27	0.045	0.67	0.90	g	g	g	g	g	g	g	g
Triglycerides	0.001	0.49	0.06	0.019	0.92	0.47	0.74	g	c,g			g		b,g	g
TC/HDL Ratio	0.011	0.81	0.39	0.08	0.12	0.61	0.89				g		g	b	g
Glucose	0.014	0.15	0.45	0.08	0.77	0.014	0.92		c		d	g			
Insulin	0.47	0.037	0.87	0.019	0.52	0.001	0.68			c			a		
HOMA	0.68	0.034	0.72	0.015	0.43	0.001	0.60			c			a		
Maximum Strength	0.001		0.35		0.38		0.67								
Bench Press Max Strength	0.001	0.07	0.67	0.22	0.10	0.19	0.27	g	g	g	d,f,g	g	g	g	g
Bench Press Lift Volume	0.96	0.60	0.64	0.39	0.30	0.024	0.57								
Leg Press Max Strength	0.001	0.047	0.30	0.46	0.26	0.16	0.40	g	g	g	d,g	g	a,g		
Leg Press Lift Volume	0.30	0.001	0.054	0.98	0.74	0.07	0.44				d	g	a	g	g
Peak VO ₂	0.001	0.043	0.054	0.001	0.40	0.85	0.13	g	c,g	c,g	f,g	g	a,g	b,g	g

Table 4.28: Continued

	Multivariate and Univariate P-Values							Post Hoc Significance							
	T	D	T*D	BP	T*BP	D*BP	T*D*BP	HP-LBP	HP-HBP	HC-LBP	HC-HBP	HP	HC	LBP	HBP
SF36 Questionnaire	0.001		0.14		0.43		0.46								
Physical Function	0.001	0.60	0.43	0.008	0.89	0.64	0.17	g	c	g	g	g	g	b,g	g
Role Physical	0.89	0.001	0.59	0.70	0.040	0.78	0.24			c	d		a	g	
Bodily Pain	0.014	0.94	0.20	0.001	0.98	0.18	0.93	g			f	g		b,g	
General Health	0.001	0.001	0.57	0.50	0.73	0.76	0.62	g	g	c,g	d,g	g	a,g	g	g
Vital	0.001	0.10	0.41	0.94	0.21	0.81	0.07	g	g	g	g	g	g	g	g
Social	0.06	0.001	0.06	0.019	0.17	0.15	0.51	g		c	d,f	g	a	g	
Role Emotional	0.630	0.001	0.07	0.79	0.70	0.96	0.69			c			a		
Mental Health	0.001	0.008	0.60	0.33	0.76	0.06	0.72	g	c,g	g	d,g	g	a,g	g	g
Body Image Questionnaire	0.001		0.54		0.88		0.77								
Appearance Evaluation	0.001	0.58	0.48	0.80	0.57	0.49	0.50	g	g	g	g	g	g	g	g
Appearance Orientation	0.32	0.043	0.55	0.007	0.68	0.34	0.53			c	f		a	b	
Body Area Satisfaction Scale	0.001	0.007	0.31	0.001	0.52	0.23	0.25	g	g	c,g	f,g	g	a,g	b,g	g
Overweight Preoccupation	0.001	0.33	0.11	0.51	0.49	0.85	0.61	g	g	g	g	g	g	g	g
Self Classified Weight	0.013	0.12	0.66	0.57	0.69	0.29	0.61								
Rosenberg Self Esteem Scale	0.006	0.54	0.17	0.37	0.34	0.70	0.42		g			g			g
Social Physique Anxiety Scale	0.61	0.95	0.98	0.70	0.61	0.45	0.84								

T = time effect, D = diet effect, T x D = time by diet effect, BP = blood pressure risk factor effect, T x BP = time by blood pressure risk factor effect, D x BP = diet by blood pressure risk factor effect, T x D x BP = time by diet by blood pressure risk factor effect. HP = higher protein, HC = higher carbohydrate, LBP = low blood pressure (<135/85 mmHg), HBP = high blood pressure (≥135/85 mmHg). N=663 participants unless stated otherwise as follows: REE N=633, Insulin and HOMA N=252, Strength N=596, SF36 N=289, and Body Image N=451.

Group breakdowns are as follows unless otherwise stated: 223 in the HP-LBP group, 148 in the HP-HBP group, 196 in the HC-LBP group, 96 in the HC-HBP group, 371 in the HP total group, 292 in the HC total group, 419 in the LBP group, 244 in the HBP group, and 663 participants total; for REE: 212 in the HP-LBP group, 145 in the HP-HBP group, 188 in the HC-LBP group, 88 in the HC-HBP group, 357 in the HP total group, 276 in the HC total group, 400 in the LBP group, 233 in the HBP group, and 633 participants total; for insulin and HOMA, 88 in the HP-LBP group, 61 in the HP-HBP group, 63 in the HC-LBP group, 40 in the HC-HBP group, 149 in the HP total group, 103 in the HC total group, 151 in the LBP group, 101 in the HBP group, and 252 participants total; for strength: 200 in the HP-LBP group, 140 in the HP-HBP group, 172 in the HC-LBP group, 84 in the HC-HBP group, 340 in the HP total group, 256 in the HC total group, 372 in the LBP group, 224 in the HBP group, and 596 participants total; for SF36: 102 in the HP-LBP group, 50 in the HP-HBP group, 95 in the HC-LBP group, 42 in the HC-HBP group, 152 in the HP total group, 137 in the HC total group, 197 in the LBP group, 92 in the HBP group, and 289 participants total; and for Body Image: 154 in the HP-LBP group, 102 in the HP-HBP group, 128 in the HC-LBP group, 67 in the HC-HBP group, 256 in the HP total group, 195 in the HC total group, 282 in the LBP group, 169 in the HBP group, and 451 participants total.

Wilks' Lambda Multivariate p-values for the group of variables are listed in bold. Greenhouse-Geisser Univariate p-values are listed for each variable included in the MANOVA (or ANOVA when indicated) analysis. Superscripts indicate significant differences between groups using Post Hoc LSD Pairwise Comparisons. ^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than HBP group, p < 0.05 (post hoc LSD). ^c Significantly different than HP-LBP group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-HBP group, p < 0.05 (post hoc LSD). ^e Significantly different than HC-LBP group, p < 0.05 (post hoc LSD). ^f Significantly different than baseline, p < 0.05 (post hoc LSD).

Post hoc analysis on the calculated delta change from baseline reveals that the HP-AH experienced a significantly greater decrease in fat percentage ($-1.7 \pm 1.9\%$) compared to HC-AH ($-1.2 \pm 2.0\%$, $p=0.018$).

Individual three-way ANOVAs were used for weight, body mass index, and waist and hip circumference. Significant univariate time x diet effects were observed for weight, body mass index, and a trend for waist circumference. The higher protein group experienced a significantly greater decrease in weight (HP -4.2 ± 3.6 , HC -3.2 ± 3.4 kg, $p<0.001$) and body mass index (HP -1.6 ± 1.3 , HC -1.2 ± 1.3 kg/m², $p<0.001$) as well as a tendency for a greater decrease in waist circumference (HP -4.0 ± 5.7 , HC -3.2 ± 5.7 cm, $p=0.095$). Both weight and body mass index experienced a trend towards a significant time x blood pressure risk factor effect. The HBP group tended to have a greater decrease in weight (LBP -3.6 ± 3.4 , HBP -4.2 ± 3.8 kg, $p=0.07$) and body mass index (LBP -1.36 ± 1.3 , HBP -1.56 ± 1.4 kg/m², $p=0.09$). While no significant time x diet x blood pressure risk factor interactions were observed, hip circumference did reveal a trend towards significance with a p-value of 0.065. Post hoc analysis on calculated delta change revealed HP-AH experienced a significantly greater decrease in hip circumference ($-3.3 \pm 4.5\%$) compared to HC-AH ($-2.3 \pm 5.0\%$, $p=0.036$). Based on these findings, hypothesis H₃₅, which stated there would be statistically significant differences observed in body composition as a result of diet intervention and hypothesis H₃₆, which stated there would be no statistically significant differences observed in body composition as a result of the presence of the blood pressure metabolic syndrome risk factor are both accepted.

Resting Energy Expenditure

The three-way ANOVA for resting energy expenditure and blood pressure risk factor revealed an overall time effect ($p < 0.001$), but no significant interactions.

Therefore, hypothesis H_{37} is accepted, which stated there would be no statistically significant differences in resting energy expenditure as a result of diet intervention and/or the presence of the blood pressure metabolic syndrome risk factor.

Resting Hemodynamic Parameters

The three-way MANOVA for resting hemodynamic parameters revealed a significant overall time (Wilks' Lambda $p < 0.001$) and time x blood pressure risk factor effect (Wilks' Lambda $p < 0.001$), but no significant time x diet (Wilks' Lambda $p = 0.48$) or time x diet x blood pressure risk factor (Wilks' Lambda $p = 0.86$) interactions. Review of the MANOVA univariate tests revealed the HBP group experienced a significantly greater *decrease* compared to an *increase* in the LBP group for both systolic (LBP $+1.1 \pm 12.3$, HBP -9.5 ± 16.1 mmHg, $p < 0.001$) and diastolic pressure, (LBP $+0.7 \pm 8.9$, HBP -6.5 ± 9.7 mmHg, $p < 0.001$). Based on these findings, hypotheses H_{38} and H_{39} are both accepted. These hypotheses stated there would be no statistically significant differences observed in resting hemodynamic parameters as a result of diet intervention, and there would be statistically significant differences observed in resting hemodynamic parameters as a result of the presence of the blood pressure metabolic syndrome risk factor, respectively.

Blood Lipids

The three-way MANOVA on blood lipids revealed a significant time (Wilks' Lambda $p < 0.001$) and time x blood pressure risk factor effect (Wilks' Lambda $p = 0.028$), but no significant time x diet (Wilks' Lambda $p = 0.40$) or time x diet x blood pressure risk factor (Wilks' Lambda $p = 0.96$) interactions. However, review of the MANOVA univariate tests does reveal a trend towards a time x diet interaction for triglycerides, which demonstrates the HP group tended to experience a greater decrease in triglyceride levels (HP -0.15 ± 0.6 , HC -0.05 ± 0.6 mmol/L, $p = 0.06$). The HBP group experienced a significantly greater decrease in HDL cholesterol (LBP -0.05 ± 0.23 , HBP -0.09 ± 0.23 mmol/L, $p = 0.045$) and tended to experience a greater decrease in total cholesterol (LBP -0.15 ± 0.78 , HBP -0.26 ± 0.85 mmol/L, $p = 0.09$). Based on these findings, hypotheses H_{40} is accepted and H_{41} is rejected. These hypotheses stated there would be statistically significant differences observed in blood lipids as a result of diet intervention, and there would be no statistically significant differences observed in blood lipids as a result of the presence of the blood pressure metabolic syndrome risk factor, respectively.

Glucose Homeostasis

The three-way ANOVA for glucose revealed an overall time effect ($p < 0.014$), but no significant interactions were observed for glucose, insulin, or HOMA. Therefore hypothesis H_{42} is rejected, which stated there would be statistically significant differences observed in markers of glucose homeostasis as a result of diet intervention. However, hypothesis H_{43} is accepted, which stated there would be no statistically

significant differences observed in markers of glucose homeostasis as a result of the presence of the blood pressure metabolic syndrome risk factor.

Fitness Parameters

The three-way MANOVA on muscular fitness revealed an overall time effect (Wilks' Lambda $p < 0.001$), but no significant time x diet (Wilks' Lambda $p = 0.35$), time x blood pressure risk factor (Wilks' Lambda $p = 0.38$), or time x diet x blood pressure risk factor (Wilks' Lambda $p = 0.67$) interactions. However, review of MANOVA univariate tests revealed a significant time x diet effect for leg press lift volume, where the higher protein group tended to have a significantly greater *increase* in leg press lift volume compared to a *decrease* in the higher carbohydrate group (HP $+111.3 \pm 938$, HC -19.9 ± 829 kg, $p = 0.054$). Additionally, a trend towards a time x blood pressure risk factor effect was observed for bench press maximum strength, in which the LBP group tended to experience a greater increase than HBP (LBP $+2.40 \pm 5.0$, HBP $+1.74 \pm 4.9$ kg, $p = 0.097$). The three-way ANOVA on cardiorespiratory fitness, measured by peak VO_2 , demonstrated a significant time ($p < 0.001$) and time x diet effect ($p = 0.054$). The higher carbohydrate group experienced a significantly greater increase in peak VO_2 (HP $+1.9 \pm 3.0$, HC $+2.6 \pm 3.7$ ml/kg/min, $p = 0.054$). Based on these findings, hypothesis H₄₄ is rejected, which stated there would be no statistically significant differences observed in fitness parameters as a result of diet intervention and/or the presence of the blood pressure metabolic syndrome risk factor.

Psychometric Analysis

The three-way MANOVA for SF36-Quality of Life demonstrated a significant time effect (Wilks' Lambda $p < 0.001$), but no significant time x diet (Wilks' Lambda $p = 0.14$), time x blood pressure risk factor (Wilks' Lambda $p = 0.43$) or time x diet x blood pressure risk factor (Wilks' Lambda $p = 0.46$) interactions. Review of the MANOVA univariate tests reveals the higher protein group tended to experience greater improvement in the social variable (HP $+4.4 \pm 15.5$, HC $+0.8 \pm 19.0$, $p = 0.056$) and the role emotional variable (HP $+20.3 \pm 81.3$, HC -9.2 ± 180.4 , $p = 0.07$). While no overall time x blood pressure risk factor effect was observed, MANOVA univariate tests revealed the LBP group experienced a significant *increase* compared to a *decrease* in the HBP group for the role physical variable (LBP $+9.4 \pm 67.9$, HBP -7.6 ± 69.2 , $p = 0.040$). A trend was also observed for time x diet x blood pressure risk factor for the vital variable ($p = 0.07$), in which post hoc analysis on the calculated delta change from baseline revealed HP-AH experienced a significantly greater increase ($+9.8 \pm 17.2$) than both HC-AH ($+4.9 \pm 13.1$, $p = 0.019$) and HP-MS ($+4.0 \pm 13.1$, $p = 0.024$).

The three-way MANOVA for Body Image demonstrated a significant time effect (Wilks' Lambda $p < 0.001$), but no significant time x diet (Wilks' Lambda $p = 0.54$), time x blood pressure risk factor (Wilks' Lambda $p = 0.88$) or time x diet x blood pressure risk factor (Wilks' Lambda $p = 0.77$) interactions. Review of the MANOVA univariate tests also did not reveal any significant interactions. Due to the significant univariate time x blood pressure interaction within the SF36 Questionnaire, hypothesis H₄₅ is rejected, which stated there would be no statistically significant differences observed in

psychometric parameters (as measured via questionnaires regarding quality of life and body image) as a result of diet intervention and/or the presence of the blood pressure metabolic syndrome risk factor.

ANALYSIS OF HYPERGLYCEMIA AS A RISK FACTOR

The hyperglycemia risk factor for metabolic syndrome in women has been defined as a fasting glucose level greater than or equal to 5.6 mmol/L. Participants have been identified as having either low glucose (LG; <5.6 mmol/L, N=390), or high glucose (HG; ≥ 5.6 mmol/L, N=273).

Energy Intake

Table 4.29 depicts the time x diet x glucose risk factor MANOVA for total energy intake in kcals/kg/day as well as macronutrient intake in g/kg/day. The three-way MANOVA revealed an overall time (Wilks' Lambda $p < 0.001$) and time x diet (Wilks' Lambda $p < 0.001$) effect, but no significant time x glucose risk factor (Wilks' Lambda $p = 0.28$) or time x diet x glucose risk factor (Wilks' Lambda $p = 0.40$) interactions. Post hoc analysis on the ten-week values was utilized to determine the following: Participants in the HC group consumed significantly more calories (HP 15.9 ± 5.2 , HC 17.4 ± 4.7 kcal/kg/d, $p < 0.001$) and carbohydrate (HP 1.41 ± 0.7 , HC 2.20 ± 0.7 g/kg/d, $p < 0.001$) relative to body weight. The HP group and the LG group consumed significantly more protein relative to body weight (HP 1.14 ± 0.5 , HC 0.78 ± 0.3 g/kg/d, $p < 0.001$), (LG 1.01 ± 0.5 , HG 0.94 ± 0.4 g/kg/d, $p = 0.014$).

Table 4.29: Dietary Intake Before and After Ten Weeks of Exercise and Dietary Intervention Based on the Hyperglycemia Risk Factor Status

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Energy Intake (kcal/kg/day)	HP-LG	20.7	± 5.8	16.0 ^g	± 5.4				T = 0.001
	HP-HG	20.0	± 5.7	15.7 ^g	± 4.7				D = 0.005
	HC-LG	21.4	± 6.7	17.7 ^{cg}	± 4.9				G = 0.06
	HC-HG	20.5	± 6.3	16.9 ^{dg}	± 4.3				D x G = 0.62
	HP	20.4	± 5.8	15.9 ^g	± 5.2	18.1 [†]	± 0.2		T x D = 0.11
	HC	21.0	± 6.6	17.4 ^{ag}	± 4.7	19.1	± 0.3		T x G = 0.72
	LG	21.0	± 6.3	16.8 ^g	± 5.3	19.0	± 0.2		T x D x G = 0.80
	HG	20.2	± 6.0	16.2 ^g	± 4.6	18.3 [‡]	± 0.3		
	Time	20.7	± 6.1	16.6 [*]	± 5.0				
Protein Intake (g/kg/day)	HP-LG	0.85	± 0.2	1.19 ^g	± 0.5				T = 0.001
	HP-HG	0.82	± 0.2	1.08 ^{cg}	± 0.4				D = 0.001
	HC-LG	0.84	± 0.2	0.80 ^c	± 0.3				G = 0.007
	HC-HG	0.81	± 0.2	0.75 ^d	± 0.2				D x G = 0.44
	HP	0.83	± 0.2	1.14 ^g	± 0.5	0.98 [†]	± 0.0		T x D = 0.001
	HC	0.83	± 0.2	0.78 ^a	± 0.3	0.80	± 0.0		T x G = 0.14
	LG	0.84	± 0.2	1.01 ^g	± 0.5	0.92	± 0.0		T x D x G = 0.42
	HG	0.81	± 0.2	0.94 ^{bg}	± 0.4	0.87 [‡]	± 0.0		
	Time	0.83	± 0.2	0.98 [*]	± 0.4				
Carbohydrate Intake (g/kg/day)	HP-LG	2.44	± 0.9	1.39 ^g	± 0.7				T = 0.001
	HP-HG	2.34	± 0.9	1.45 ^g	± 0.7				D = 0.001
	HC-LG	2.56	± 1.0	2.45 ^{cg}	± 0.7				G = 0.24
	HC-HG	2.47	± 0.9	2.14 ^{dg}	± 0.6				D x G = 0.41
	HP	2.40	± 0.9	1.41 ^g	± 0.7	1.91 [†]	± 0.0		T x D = 0.001
	HC	2.52	± 0.9	2.20 ^{ag}	± 0.7	2.35	± 0.0		T x G = 0.31
	LG	2.50	± 0.9	1.77 ^g	± 0.8	2.16	± 0.0		T x D x G = 0.24
	HG	2.40	± 0.9	1.74 ^g	± 0.7	2.10	± 0.0		
	Time	2.46	± 0.9	1.76 [*]	± 0.8				
Fat Intake (g/kg/day)	HP-LG	0.84	± 0.3	0.64 ^g	± 0.3				T = 0.001
	HP-HG	0.82	± 0.3	0.62 ^g	± 0.2				D = 0.70
	HC-LG	0.86	± 0.3	0.61 ^g	± 0.2				G = 0.17
	HC-HG	0.82	± 0.3	0.59 ^g	± 0.2				D x G = 0.63
	HP	0.83	± 0.3	0.63 ^g	± 0.3	0.73	± 0.0		T x D = 0.13
	HC	0.85	± 0.3	0.60 ^g	± 0.2	0.72	± 0.0		T x G = 0.75
	LG	0.85	± 0.3	0.63 ^g	± 0.3	0.74	± 0.0		T x D x G = 0.64
	HG	0.82	± 0.3	0.61 ^g	± 0.2	0.72	± 0.0		
	Time	0.84	± 0.3	0.62 [*]	± 0.2				

HP = higher protein, HC = higher carbohydrate, LG = low glucose, MS = high glucose risk factor, T = time effect, D = diet effect, G = glucose risk factor effect, D x G = diet by glucose risk factor effect, T x D = time by diet effect, T x G = time by glucose risk factor effect, T x D x G = time by diet by glucose risk factor effect.

Values are means ± standard deviations (except group means are means ± standard error) from 214 participants in the HP-LG group, 157 in the HP-HG group, 176 in the HC-LG group, 116 in the HC-HG group, 371 in the HP total group, 292 in the HC total group, 390 in the LG group, 273 in the HG group, and 663 participants total.

* Significantly different than baseline, $p < 0.05$ (univariate). † Significant diet effect, $p < 0.05$ (univariate). ‡ Significant glucose effect, $p < 0.05$ (univariate). ^a Significantly different than HP group, $p < 0.05$ (post hoc LSD). ^b Significantly different than LG group, $p < 0.05$ (post hoc LSD). ^c Significantly different than HP-LG group, $p < 0.05$ (post hoc LSD). ^d Significantly different than HP-HG group, $p < 0.05$ (post hoc LSD). ^e Significantly different than HC-LG group, $p < 0.05$ (post hoc LSD). ^g Significantly different than baseline, $p < 0.05$ (post hoc LSD).

Regarding specific group-combinations, energy intake was significantly greater for HC-LG compared to HP-LG ($p=0.001$), and HC-HG was significantly greater than HP-HG ($p=0.050$). For carbohydrate intake, HC-LG was significantly greater than HP-LG ($p<0.001$), and HC-HG was significantly greater than HP-HG ($p<0.001$). For protein intake, HP-LG was significantly greater than both HP-HG ($p=0.011$) and HC-LG ($p<0.001$), and HP-HG was significantly greater than HC-HG ($p<0.001$). No significant differences were observed between group-combinations regarding fat intake.

Overall Analysis

Table 4.30 represents the Greenhouse-Geisser univariate and (when applicable) Wilks' Lambda multivariate p-levels for each of the variables and groups measured and analyzed by the various diet and glucose risk factor interactions (time, diet, glucose risk factor, time x diet, time x glucose risk factor, diet x glucose risk factor, and time x diet x glucose risk factor). The significant findings per variable are discussed below. When a significant three-way interaction was observed, the significant group differences revealed in the post hoc LSD pairwise comparisons analysis are also indicated. Each corresponding hypothesis is also evaluated.

Body Composition

Within the body composition analyses, a significant time (Wilks' Lambda $p<0.001$) and time x diet effect (Wilks' Lambda $p=0.030$) was observed for the DEXA variables, but no significant time x glucose risk factor (Wilks' Lambda $p=0.74$) or time x diet x glucose risk factor (Wilks' Lambda $p=0.30$) interactions were observed.

Table 4.30: Significance Levels for Glucose Risk Factor Analysis per Variable

	Multivariate and Univariate P-Values							Post Hoc Significance							
	T	D	T*D	G	T*G	D*G	T*D*G	HP-LG	HP-HG	HC-LG	HC-HG	HP	HC	LG	HG
Body Composition	0.001		0.030		0.74		0.30								
Scanned Mass	0.001	0.001	0.001	0.002	0.47	0.99	0.88	g	c,g	c,g	d,f,g	g	a,g	b,g	g
Fat Mass	0.001	0.001	0.004	0.006	0.23	0.99	0.27	g	c,g	c,g	d,g	g	a,g	b,g	g
Lean Mass	0.001	0.001	0.12	0.002	0.68	0.81	0.23	g	c,g	c,g	d,f,g	g	a,g	b,g	g
Body Fat	0.001	0.055	0.28	0.14	0.18	0.84	0.056	g	g	g	g	g	g	g	g
Weight	0.001	0.001	0.001	0.001	0.32	0.82	0.44	g	c,g	c,g	d,f,g	g	a,g	b,g	g
Body Mass Index	0.001	0.001	0.001	0.001	0.32	0.68	0.47	g	c,g	c,g	d,f,g	g	a,g	b,g	g
Waist Circumference	0.001	0.001	0.14	0.001	0.90	0.92	0.07	g	c,g	c,g	d,f,g	g	a,g	b,g	g
Hip Circumference	0.001	0.001	0.38	0.001	0.63	0.62	0.033	g	c,g	c,g	d,f,g	g	a,g	b,g	g
Resting Energy Expenditure	0.001	0.001	0.39	0.001	0.051	0.65	0.46	g	c,g	c	d,g	g	a,g	b,g	g
Hemodynamic	0.001		0.89		0.47		0.025								
Resting Heart Rate	0.001	0.21	0.79	0.019	1.000	0.20	0.78	g	c,g	g	g	g	g	b,g	g
Systolic Blood Pressure	0.001	0.13	0.92	0.001	0.49	0.77	0.002		g	c,g	f	g	g	b,g	g
Diastolic Blood Pressure	0.001	0.001	0.55	0.001	0.11	0.74	0.15		g	c,g	f,g	g	a,g	b,g	g
Blood Lipids	0.001		0.65		0.001		0.048								
Total Cholesterol	0.001	0.72	0.47	0.001	0.001	0.54	0.77		g		g	g	g	g	g
LDL Cholesterol	0.001	0.55	0.66	0.001	0.001	0.41	0.57		c,g		g	g	g	b,g	g
HDL Cholesterol	0.001	0.72	0.96	0.001	0.011	0.48	0.27	g	c,g	g	f,g	g	g	b,g	g
Triglycerides	0.001	0.45	0.09	0.001	0.09	0.21	0.14	g	c,g		f,g	g		b,g	g
TC/HDL Ratio	0.025	0.93	0.42	0.001	0.70	0.20	0.68		c		f		g	b	
Glucose	0.001	0.32	0.81	0.001	0.001	0.07	0.017		c,g	g	d,f,g	g	g	b,g	g
Insulin	0.74	0.002	0.59	0.001	0.45	0.38	0.36		c		f		a	b	
HOMA	0.94	0.002	0.38	0.001	0.28	0.19	0.19		c		f		a	b	
Maximum Strength	0.001		0.40		0.54		0.48								
Bench Press Max Strength	0.001	0.15	0.41	0.13	0.34	0.96	0.88	g	g	g	g	g	g	g	g
Bench Press Lift Volume	0.76	0.82	0.55	0.38	0.78	0.73	0.45								
Leg Press Max Strength	0.001	0.10	0.32	0.90	0.77	0.67	0.15	g	g	g	g	g	g	g	g
Leg Press Lift Volume	0.15	0.003	0.11	0.36	0.21	0.68	0.50			c		g	a		
Peak VO ₂	0.001	0.020	0.026	0.001	0.014	0.85	0.28	g	c,g	c,g	f,g	g	a,g	b,g	g

Table 4.30: Continued

	Multivariate and Univariate P-Values							Post Hoc Significance							
	T	D	T*D	G	T*G	D*G	T*D*G	HP-LG	HP-HG	HC-LG	HC-HG	HP	HC	LG	HG
SF36 Questionnaire	0.001		0.12		0.42		0.25								
Physical Function	0.001	0.62	0.65	0.20	0.79	0.38	0.30	g	c	g	g	g	g	g	g
Role Physical	0.43	0.001	0.95	0.16	0.13	0.61	0.51			c	d		a		
Bodily Pain	0.009	0.50	0.18	0.25	0.81	0.69	0.94	g	g			g			
General Health	0.001	0.001	0.53	0.03	0.19	0.79	0.10	g	c	c.g	d.g	g	a.g	b.g	g
Vital	0.001	0.09	0.24	0.97	0.37	0.33	0.020	g	g		g	g	g	g	g
Social	0.018	0.001	0.08	0.27	0.10	0.24	0.51	g	c		d	g	a	g	
Role Emotional	0.64	0.001	0.056	0.50	0.19	0.64	0.74			c	d		a		
Mental Health	0.001	0.036	0.71	0.12	0.54	0.57	0.014	g	c.g	g	d.g	g	g	g	g
Body Image Questionnaire	0.001		0.62		0.55		0.041								
Appearance Evaluation	0.001	0.54	0.64	0.65	0.08	0.56	0.57	g	g	g	g	g	g	g	g
Appearance Orientation	0.31	0.032	0.66	0.002	0.52	0.28	0.15		c	c			a	b	
Body Area Satisfaction Scale	0.001	0.002	0.32	0.18	0.56	0.69	0.25	g	g	c.g	g	g	a.g	g	g
Overweight Preoccupation	0.001	0.22	0.26	0.31	0.72	0.29	0.18	g	g	g	g	g	g	g	g
Self Classified Weight	0.008	0.036	0.93	0.009	0.41	0.35	0.010		g	c.g		g			g
Rosenberg Self Esteem Scale	0.006	0.67	0.10	0.055	0.53	0.62	0.11		g			g			g
Social Physique Anxiety Scale	0.74	0.82	0.70	0.72	0.95	0.99	0.18								

T = time effect, D = diet effect, T x D = time by diet effect, G = glucose risk factor effect, T x G = time by glucose risk factor effect, D x G = diet by glucose risk factor effect, T x D x G = time by diet by glucose risk factor effect. HP = higher protein, HC = higher carbohydrate, LG = low glucose (<5.6 mmol/L), HG = high glucose (≥5.6 mmol/L). N=663 participants unless stated otherwise as follows: REE N=633, Insulin and HOMA N=252, Strength N=596, SF36 N=289, and Body Image N=451. Group breakdowns are as follows unless otherwise stated: 214 in the HP-LG group, 157 in the HP-HG group, 176 in the HC-LG group, 116 in the HC-HG group, 371 in the HP total group, 292 in the HC total group, 390 in the LG group, 273 in the HG group, and 663 participants total; for REE: 209 in the HP-LG group, 148 in the HP-HG group, 169 in the HC-LG group, 107 in the HC-HG group, 357 in the HP total group, 276 in the HC total group, 378 in the LG group, 255 in the HG group, and 633 participants total; for insulin and HOMA: 109 in the HP-LG group, 40 in the HP-HG group, 67 in the HC-LG group, 36 in the HC-HG group, 149 in the HP total group, 103 in the HC total group, 176 in the LG group, 76 in the HG group, and 252 participants total; for strength: 196 in the HP-LG group, 144 in the HP-HG group, 154 in the HC-LG group, 102 in the HC-HG group, 340 in the HP total group, 256 in the HC total group, 350 in the LG group, 246 in the HG group, and 596 participants total; for SF36: 83 in the HP-LG group, 69 in the HP-HG group, 82 in the HC-LG group, 55 in the HC-HG group, 152 in the HP total group, 137 in the HC total group, 165 in the LG group, 124 in the HG group, and 289 participants total; and for Body Image: 161 in the HP-LG group, 95 in the HP-HG group, 120 in the HC-LG group, 75 in the HC-HG group, 256 in the HP total group, 195 in the HC total group, 281 in the LG group, 170 in the HG group, and 451 participants total. Wilks' Lambda Multivariate p-values for the group of variables are listed in bold. Greenhouse-Geisser Univariate p-values are listed for each variable included in the MANOVA (or ANOVA when indicated) analysis. Superscripts indicate significant differences between groups using Post Hoc LSD Pairwise Comparisons. ^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than HG group, p < 0.05 (post hoc LSD). ^c Significantly different than HP-LG group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-HG group, p < 0.05 (post hoc LSD). ^e Significantly different than HC-LG group, p < 0.05 (post hoc LSD). ^f Significantly different than baseline, p < 0.05 (post hoc LSD).

Review of the MANOVA univariate tests revealed the higher protein group experienced a significantly greater decrease for both scanned mass (HP -3.9 ± 3.5 , HC -3.0 ± 3.5 kg, $p=0.001$) and fat mass (HP -3.1 ± 2.7 , HC -2.4 ± 2.8 kg, $p=0.004$). Additionally, a trend was observed for body fat for the time x diet x glucose risk factor interaction ($p=0.056$). Post hoc analysis of body composition data expressed in delta changes from baseline revealed the HC-LG group experienced a significantly smaller decrease ($-1.16 \pm 2.0\%$) in fat percentage than both HP-LG ($-1.62 \pm 1.8\%$, $p=0.020$) and HC-HG ($-1.66 \pm 1.9\%$, $p=0.031$).

Individual three-way ANOVAs were used for weight, body mass index, and waist and hip circumference. Significant time x diet effects were observed for both weight and body mass index. The higher protein group experienced a significantly greater decrease in both weight (HP -4.3 ± 3.6 , HC -3.2 ± 3.4 kg, $p<0.001$) and body mass index (HP -1.6 ± 1.3 , HC -1.2 ± 1.3 kg/m², $p<0.001$). Additionally, a significant three-way interaction on time x diet x glucose risk factor was observed for hip circumference ($p=0.033$) and a trend was observed for waist circumference ($p=0.073$). Post hoc analysis on waist and hip circumference as delta change from baseline revealed the HP-LG experienced a significantly greater decrease than HC-LG in waist circumference (HP-LG -4.3 ± 5.5 , HC-LG -2.8 ± 5.7 cm, $p=0.010$) and hip circumference (HP-LG -3.5 ± 5.1 , HC-LG -2.3 ± 5.1 cm, $p=0.019$). Based on these findings, hypothesis H₄₆ which stated there would be statistically significant differences observed in body composition as a result of diet intervention, is accepted, and hypothesis H₄₇ which stated there would

be no statistically significant differences observed in body composition as a result of the presence of the glucose metabolic syndrome risk factor, is rejected.

Resting Energy Expenditure

The three-way ANOVA on resting energy expenditure revealed a significant time ($p<0.001$) and time x glucose risk factor effect ($p=0.051$). Participants in the high glucose group experienced a significantly greater decrease in resting energy expenditure (LG -42.4 ± 202.7 , HG -75.0 ± 224 kcal/d, $p=0.051$). Based on this finding, hypothesis H₄₈ is rejected, which stated there would be no statistically significant differences observed in resting energy expenditure parameters as a result of diet intervention and/or the presence of the glucose metabolic syndrome risk factor.

Resting Hemodynamic Parameters

No significant overall time x diet (Wilks' Lambda $p=0.89$) or time x glucose risk factor (Wilks' Lambda $p=0.47$) effects were observed within the MANOVA on resting hemodynamic parameters, however significant time (Wilks' Lambda $p<0.001$) and time x diet x glucose risk factor (Wilks' Lambda $p=0.025$) effects were observed. Post hoc analysis using calculations of delta change from baseline revealed the HC-HG group experienced a significantly greater decrease in systolic blood pressure than all other group-combination (-5.1 ± 14.9 mmHg, $p=0.040$). Based on this finding, hypothesis H₄₉ is rejected, which stated there would be no statistically significant differences observed in resting hemodynamic parameters as a result of diet intervention and/or the presence of the glucose metabolic syndrome risk factor.

Blood Lipids

The three-way MANOVA on blood lipid variables revealed a significant time (Wilks' Lambda $p<0.001$), time x glucose risk factor (Wilks' Lambda $p<0.001$), and time x diet x glucose risk factor (Wilks' Lambda $p=0.048$) effect, but no significant time x diet (Wilks' Lambda $p=0.65$) interaction. While the overall MANOVA time x diet interaction was not significant, review of MANOVA univariate tests reveals that the higher protein group tended to experience a greater decrease in triglyceride levels (HP -0.15 ± 0.62 , HC -0.05 ± 0.60 mmol/L, $p=0.09$). Participants with high glucose experienced significantly greater decreases in total cholesterol (LG -0.09 ± 0.8 , HG -0.33 ± 0.9 mmol/L, $p<0.001$), LDL cholesterol (LG -0.07 ± 0.6 , HG -0.25 ± 0.7 mmol/L, $p<0.001$), and a tendency for a greater decrease in triglycerides (LG -0.07 ± 0.5 , HG -0.14 ± 0.7 mmol/L, $p=0.094$). Participants in the high glucose group also experienced a significantly greater decrease in HDL cholesterol (LG -0.05 ± 0.24 , HG -0.09 ± 0.2 mmol/L, $p=0.011$). While the three-way MANOVA on blood lipids demonstrated a trend towards significance for time x diet x glucose risk factor, when each of the lipid variables were analyzed independently, the interaction did not prove significant. Based on these findings, hypothesis H₅₀, which stated there would be statistically significant differences observed in blood lipids as a result of diet intervention and/or the presence of the glucose metabolic syndrome risk factor, is accepted.

Glucose Homeostasis

The three-way ANOVA on glucose revealed a significant time ($p<0.001$), time x glucose risk factor ($p<0.001$) as well as a time x diet x glucose risk factor ($p=0.017$)

effect. Participants with high glucose levels experienced a significantly greater *decrease* in fasting glucose in comparison to a mild *increase* in the low glucose group (LG $+0.12 \pm 0.6$, HG -0.4 ± 1.3 mmol/L, $p < 0.001$). Post hoc analysis on glucose data expressed in delta changes from baseline revealed the HP-HG group experienced a significantly greater *decrease* in fasting glucose (-0.34 ± 1.3 mmol/L) compared to a slight *increase* in the HP-LG group ($+0.03 \pm 0.6$ mmol/L, $p < 0.001$), and likewise the HC-HG group experienced a significantly greater *decrease* (-0.49 ± 1.2 mmol/L) compared to a slight *increase* in the HC-LG group ($+0.22 \pm 0.5$ mmol/L, $p < 0.001$). No significant effects were observed within the insulin or HOMA analyses. Based on these findings hypothesis H₅₁ is accepted, which stated there would be statistically significant differences observed in markers of glucose homeostasis as a result of diet intervention and/or the presence of the glucose metabolic syndrome risk factor.

Fitness Parameters

No significant interactions were observed in the three-way MANOVA on muscular fitness parameters, however there was a significant time effect (Wilks' Lambda $p < 0.001$). The ANOVA on cardiorespiratory fitness, measured by peak VO₂, demonstrated a significant time (Greenhouse-Geisser $p < 0.001$), time x diet (Greenhouse-Geisser $p = 0.026$) and a time x glucose risk factor (Greenhouse-Geisser $p = 0.014$) effect. The higher carbohydrate and low glucose groups both experienced a significantly greater increase in peak VO₂ (HP $+1.9 \pm 3.0$, HC $+2.6 \pm 3.7$ ml/kg/min, $p = 0.026$), (LG $+2.5 \pm 3.6$, HG $+1.8 \pm 3.0$ ml/kg/min, $p = 0.014$). Based on these results, hypothesis H₅₂ which stated there would be no statistically significant differences observed in fitness parameters as a

result of diet intervention and/or the presence of the glucose metabolic syndrome risk factor, is rejected.

Psychometric Analysis

The three-way MANOVA on SF36-Quality of Life Questionnaire revealed a significant time (Wilks' Lambda $p < 0.001$), but no significant time x diet (Wilks' Lambda $p = 0.12$), time x glucose risk factor (Wilks' Lambda $p = 0.42$), or time x diet x glucose risk factor (Wilks' Lambda $p = 0.25$) interactions. However, review of the MANOVA univariate tests revealed trends towards significance for both the social and role emotional variables for time x diet. There was a tendency for a greater increase in the HP group for both the social aspect (HP $+4.5 \pm 15.5$, HC $+0.9 \pm 19.2$, $p = 0.08$) and the role emotional aspect (HP $+20.3 \pm 81.3$, HC -9.2 ± 180.4 , $p = 0.056$). The social aspect also experienced a trend for time x glucose risk factor, where the LG group tended to experience a greater increase (LG $+4.2 \pm 15.6$, HG $+0.9 \pm 19.0$, $p = 0.10$). Finally, for the time x diet x glucose risk factor interaction, the general health aspect experienced a trend ($p = 0.10$), and both the vital aspect ($p = 0.02$) and the mental health aspect ($p = 0.014$) were significant. Post hoc analysis on the delta changes from baseline revealed that the HP-LG group experienced a significantly greater increase in the general health aspect than HP-HG (HP-LG $+6.3 \pm 13.0$, HP-HG $+2.1 \pm 10.6$, $p = 0.030$). Regarding the vital aspect, the HC-LG group experienced significantly less of an increase ($+2.9 \pm 12.7$) compared to both HP-LG ($+9.0 \pm 12.4$, $p = 0.007$) and HC-HG ($+8.6 \pm 12.7$, $p = 0.27$). For mental health, the HP-LG group experienced a significantly greater increase ($+11.1 \pm 13.7$) compared to both the HP-HG ($+5.6 \pm 19.4$, $p = 0.024$) and HC-LG ($+6.0 \pm 12.0$, $p = 0.029$) groups.

The three-way MANOVA for the Body Image Questionnaire variables revealed a significant time (Wilks' Lambda $p < 0.001$) and time x diet x glucose risk factor (Wilks' Lambda $p = 0.041$) effect, but no significant time x diet (Wilks' Lambda $p = 0.62$) or time x glucose risk factor (Wilks' Lambda $p = 0.55$) interactions. However, review of the MANOVA univariate tests revealed a trend towards significance for time x diet for the Rosenberg self esteem scale, where the HP group tended to experience a greater increase (HP $+0.6 \pm 3.2$, HC $+0.2 \pm 3.4$, $p = 0.10$). Additionally, a trend towards significance for time x glucose risk factor was observed for appearance evaluation, where the LG group tended to experience a greater increase (LG $+0.39 \pm 0.6$, HG $+0.29 \pm 0.6$, $p = 0.08$).

Regarding the significant overall three-way interaction, self-classified weight revealed the significant univariate interaction ($p = 0.010$). Post hoc analysis using the delta changes from baseline revealed that for the self-classified weight variable the HP-LG group experienced a significant *increase* ($+0.02 \pm 0.8$) compared to *decreases* in both HP-HG (-0.24 ± 0.6 , $p = 0.010$) and HC-LG (-0.17 ± 0.7 , $p = 0.044$). Based on these findings, hypothesis H₅₃ is rejected, which stated there would be no statistically significant differences observed in psychometric assessments (as measured via questionnaires regarding quality of life and body image) as a result of diet intervention and/or the presence of the glucose metabolic syndrome risk factor.

CHAPTER V

SUMMARY

This analysis sought to determine if following a higher protein diet intervention for ten-weeks promotes a reduction in metabolic syndrome and the individual NCEP ATP III risk factors more effectively than a higher carbohydrate diet for the same time period, when combined with a circuit-style resistance-based program. Overall, participants in both dietary groups decreased body fat, resting heart rate and blood pressure, improved lipid profiles, and increased strength with no significant differences observed between groups. Approximately 80% of weight lost for all participants was fat loss. However, participants following the higher protein diet experienced a greater improvement in body composition (scanned mass, fat mass, and weight, with a trend for waist circumference). Individuals classified as having metabolic syndrome benefited from greater reductions in body composition, blood pressure, triglycerides and glucose than participants categorized as apparently healthy, and participants with metabolic syndrome on the higher protein diet (HP-MS) experienced the greatest decrease in scanned mass overall. Generally, participants with metabolic syndrome were more overweight, had higher blood pressure, and had less optimal lipid profiles at the beginning of the study, and therefore achieved greater benefits at the end of the ten-week diet and exercise protocol. Similar results were found for each of the individual risk factor stratifications, in which the groups with elevated risk factors experienced greater benefits in that variable over the ten-week term.

This analysis generally concurs with earlier research on dietary interventions with, and without, exercise prescriptions. Previous studies have demonstrated that a higher protein diet can lead to a greater reduction in weight [131, 134, 135, 139, 142, 144, 145] and fat mass [43, 63, 120, 122, 125, 131, 142, 146, 147, 180, 203], when compared to a higher carbohydrate diet alone and also when combined with exercise [42, 43, 172, 174, 178-180]. This analysis also agrees with other studies, specific to metabolic syndrome risk factors, that have shown no significant difference between higher carbohydrate and higher protein diets but observed an overall decrease in blood pressure [42, 67, 120, 131, 132, 135-137, 145, 152, 153, 156, 174, 175, 181, 236], HDL cholesterol [42, 67, 133, 153, 154, 173], and glucose [42, 131, 132, 136-139, 142, 147, 148, 154, 156, 174, 236, 237].

Many short-term studies comparing differing carbohydrate and protein allotments without an exercise component report findings similar to the present analysis. For example, Morenga et al[142] evaluated the effect of a moderate-high protein consumption (PRO:CHO:FAT 30:40:30) versus a high carbohydrate diet (20:50:30) on obese women for an eight-week period and reported that both groups lost weight, body fat, waist circumference, blood pressure, total cholesterol, LDL cholesterol, and glucose. Similar to the present analysis, the higher protein group lost significantly more weight and total body fat, yet Morenga's participants also experienced a greater decrease in diastolic blood pressure. Additionally, a twelve-week study by Noakes et al[173] compared obese females consuming either a higher protein (34:46:20) or a higher carbohydrate (17:64:20) diet and similarly revealed a decrease in weight, glucose, and

HDL cholesterol for both groups, yet also found a significantly greater decrease in triglycerides for the higher protein group. This study also categorized participants based on triglyceride level ($<$ or >1.5 mmol/L) and, similar to the present analysis, found that subjects with higher triglycerides lost a greater amount of fat mass on the higher protein diet.

Furthermore, similar findings have been reported by many short-term studies with dietary assignments comparable to the present analysis that also include an exercise component. In a four-month study by Layman et al[43], evaluating obese females consuming either a higher carbohydrate (0.8 g/kg/d PRO, 3.5 g/kg/d CHO) or a higher protein (1.6 g/kg/d PRO, <1.5 g/kg/d CHO) diet while participating in either voluntary walking or mandatory resistance training, the higher protein groups (regardless of exercise intervention) lost significantly more weight and fat mass, as observed in the present analysis, yet also experienced greater reductions in triglycerides. Additionally, Layman et al also observed better maintenance of HDL cholesterol in their higher protein group and greater reductions in total and LDL cholesterol in their carbohydrate group, which were not reported in the present analysis. Meckling and Sherfey[120] conducted a twelve-week study measuring the change in MetS risk factors in overweight females by utilizing a control group (CHO:PRO = 3.0) versus a higher protein (CHO:PRO = 1.0) diet with or without a 36-minute circuit training program three times a week. Similar to the present analysis, exercising participants in the Meckling study improved cardiovascular fitness and lost weight (although this study found no differences based on diet), yet the higher protein diet combined with exercise lead to a

greater loss in triglycerides. In a sixteen-week study by Wycherley et al[42], obese subjects with type 2 diabetes consumed either a higher carbohydrate (19:53:26) diet, or a higher protein diet (33:43:22), with or without the addition of resistance training three times a week. While all groups improved body composition, blood pressure, lipid profile, and insulin sensitivity, the higher protein group with resistance training lost a significantly greater amount of weight, fat mass, and waist circumference, as was the case in the present analysis. Finally, Layman et al[180] performed a four-month study on obese subjects comparing a higher carbohydrate (15:55:30; 0.8 g/kg/d PRO) and a higher protein (30:40:30; 1.6 g/kg/d PRO) diet with thirty-minutes of walking five times a week. Similar to the present analysis, they found a greater decrease in fat mass in the HP group, however they also reported a greater decrease in triglycerides in the HP group and in total and LDL cholesterol in the HC group. Interestingly, the beneficial affects of the HP diet on body composition and triglyceride levels were sustained during an eight-month follow up, while the changes to the lipid profile based on the HC diet were not. This is promising for the results presented for the higher protein group in the present analysis.

Many studies have shown diet and exercise protocols to be effective for subjects specifically with metabolic syndrome as well. Miller et al[89] utilized a high protein and high fat diet (30:27:43) for three months in adults with MetS and observed a significant decrease in weight, waist circumference, blood pressure, and the prevalence of MetS. In a six-week study on MetS subjects by Rajaie et al[111], a moderately-restricted carbohydrate diet lead to significantly greater reductions in diastolic blood pressure and

MetS prevalence. Lopez-Jimenez et al [148] did not observe much difference between a low fat diet and a lower carbohydrate / higher protein diet in a six month protocol on metabolic syndrome, but observed an overall decrease in weight, triglycerides and glucose, with a greater decrease in MetS prevalence in the lower carbohydrate group. In a twelve-month study on MetS subjects by Flechtner-Mors et al[139], the higher protein group (30:40:30) experienced significantly greater reductions in weight, waist circumference, triglycerides, and MetS prevalence. Finally, similar to the present analysis, a study by Campbell and Meckling[67] that incorporated a 60-minute exercise circuit three times a week in addition to numerous protein:carbohydrate ratios observed few significant differences between the dietary protocols, but an overall decrease in weight, waist circumference, blood pressure, and triglycerides. These studies support the present analysis in their findings that regardless of the specific dietary protocol, diet and exercise can lead to a reduction in prevalence of metabolic syndrome and its individual risk factors.

Several studies, however, have reported findings that were not supported by the present analysis. Numerous previous observations have reported that replacing some carbohydrate consumption with protein can lead to a greater decrease in triglyceride levels through diet alone [31-33, 122-139] and when combined with exercise [43, 120, 173, 174, 178-180, 182], which was not observed in the present analysis. Some studies have stated that higher protein intake more effectively leads to improved lipid profile and glycemic control [43, 63, 120], yet this observation was not apparent in the present analysis. However, these studies suggest a protein intake >1.4 g/kg/day, and the present

analysis only utilized an intake of 1.14 g/kg/day protein for the HP group. Other studies have indicated higher protein diets alone [123, 126, 131, 132, 134-136, 139] and combined with exercise [172, 175, 178-181] are more effective at increasing HDL cholesterol. Conversely, the present analysis experienced a decrease in HDL throughout the study with no difference between diet groups, yet a larger decrease in the apparently healthy group than the metabolic syndrome group. However, overall the AH group mean HDL was 1.4 ± 0.3 mmol/L at the end of the study, well above the <1.3 mmol/L cut off point that defines HDL as a MetS risk factor. Additionally, evidence suggests that lower fat diets may be responsible for the decrease in HDL cholesterol [97, 238], and/or that HDL particle size may shift during weight loss [238-240], but also that this decrease in HDL during weight loss may increase again once weight is stabilized [58]. The aforementioned shift is not taken into consideration within the HDL lipid measurement and therefore these results may not actually be indicative of an increase in risk, particularly considering total cholesterol, LDL cholesterol and triglycerides all decreased in the present analysis without a change in TC/HDL or TG/HDL ratios.

CONCLUSION

Overall, the present analysis has shown that diet and exercise are beneficial for improving markers of health. This retrospective analysis was partially based on recommendations by Layman et al [43], suggesting that a CHO-based, low-fat diet may work better for persons needing to lower total cholesterol and LDL, while a PRO-based diet could positively benefit individuals who have elevated TG and low HDL. Unfortunately, the present analysis did not indicate a differential effect in blood lipid

values based on macronutrient ratios. However, beneficial effects on body composition were seen with the higher protein diet regardless of whether individuals were stratified based on general metabolic syndrome status or by each MetS risk factor individually. The macronutrient ratios utilized for the higher carbohydrate group in this analysis correspond to the NCEP ATP III recommendations for individuals with MetS [17], and the protein intake in the HC group matches the current recommended daily allowance for protein [114, 115]. However the results from this analysis indicate that the current RDA for protein intake in the general population, as well as the dietary recommendations set forth by the NCEP ATP III for individuals with metabolic syndrome may not be the most effective, and should be replaced with a higher protein requirement in order to more effectively alter body composition in subjects with metabolic syndrome.

Due to the nature of a retrospective analysis, this study experienced a few limitations and restrictions regarding the analysis and information available within the dataset. One limitation regarding the analysis was that participants were assigned to a dietary protocol based on their response to the Carbohydrate Intolerance Questionnaire (Appendix F). The fact that participants were initially labeled as carbohydrate tolerant or intolerant, and placed into higher carbohydrate or higher protein diets respectively, may produce a confounding variable within the analysis. However, this questionnaire is an integral part of the Curves program that was utilized within the eight studies analyzed, and therefore unavoidable for the present analysis. Moving forward, it would be beneficial to reproduce a similar analysis with participants randomly assigned to the dietary protocols and also for future research using these risk factor classifications to

include more specific dietary analysis (such as categorizing carbohydrate consumption as simple or complex, and fat consumption as saturated or unsaturated). Additionally, the ability to analyze the population based on ethnic affiliation or menopause status may also reveal beneficial findings.

REFERENCES

1. Grundy SM, Cleeman JJ, Daniels SR, Donato KA, Eckel RH, Franklin BA, Gordon DJ, Krauss RM, Savage PJ, Smith Jr SC: **Diagnosis and management of the metabolic syndrome.** *Circulation* 2005, **112**(17):2735-2752.
2. Lakka HM, Laaksonen DE, Lakka TA, Niskanen LK, Kumpusalo E, Tuomilehto J, Salonen JT: **The metabolic syndrome and total and cardiovascular disease mortality in middle-aged men.** *JAMA: The Journal of the American Medical Association* 2002, **288**(21):2709-2716.
3. Meigs JB: **Metabolic syndrome: is there treatment that works?** *Contemporary Endocrinology: Controversies in Treating Diabetes: Clinical and Research Aspects.* Edited by LeRoith D, Vinik AI. Totowa, NJ: Humana Press; 2008:33-50.
4. Wilson PWF, D'Agostino RB, Parise H, Sullivan L, Meigs JB: **Metabolic syndrome as a precursor of cardiovascular disease and type 2 diabetes mellitus.** *Circulation* 2005, **112**(20):3066-3072.
5. Eriksson J, Taimela S, Koivisto V: **Exercise and the metabolic syndrome.** *Diabetologia* 1997, **40**(2):125-135.
6. Ford ES, Li C: **Physical activity or fitness and the metabolic syndrome.** *Expert Review of Cardiovascular Therapy* 2006, **4**(6):897-915.
7. Alberti K: **Metabolic syndrome - a new world-wide definition. A consensus statement from the International Diabetes Federation.** *Diabetic Medicine* 2006, **23**(5):469-480.
8. Eckel RH, Grundy SM, Zimmet PZ: **The metabolic syndrome.** *Lancet* 2005, **365**(9468):1415-1428.
9. Ford ES, Giles WH: **A comparison of the prevalence of the metabolic syndrome using two proposed definitions.** *Diabetes Care* 2003, **26**(3):575-581.
10. Meigs JB, Wilson PWF, Nathan DM, D'Agostino RB, Williams K, Haffner SM: **Prevalence and characteristics of the metabolic syndrome in the San Antonio Heart and Framingham Offspring Studies.** *Diabetes* 2003, **52**(8):2160-2167.
11. Hunt KJ, Resendez RG, Williams K, Haffner SM, Stern MP: **National Cholesterol Education Program versus World Health Organization metabolic syndrome in relation to all-cause and cardiovascular mortality in the San Antonio Heart Study.** *Circulation* 2004, **110**(10):1251-1257.

12. Grundy SM, Cleeman JI, Merz CNB, Brewer HB, Clark LT, Hunninghake DB, Pasternak RC, Smith SC, Stone NJ: **Implications of recent clinical trials for the national cholesterol education program adult treatment panel III guidelines.** J Am Coll Cardiol 2004, **44**(3):720-732.
13. Tjønnå AE, Lee SJ, Rognmo Ø, Stølen TO, Bye A, Haram PM, Loennechen JP, Al-Shaie QY, Skogvoll E, Slørdahl SA, Kemi OJ, Najjar SM, Wisløff U: **Aerobic interval training versus continuous moderate exercise as a treatment for the metabolic syndrome.** Circulation 2008, **118**(4):346-354.
14. Isomaa B: **A major health hazard: the metabolic syndrome.** Life Sci 2003, **73**(19):2395.
15. Grundy SM, Brewer Jr HB, Cleeman JI, Smith Jr SC, Lenfant C: **Definition of metabolic syndrome report of the National Heart, Lung, and Blood Institute/American Heart Association Conference on scientific issues related to definition.** Circulation 2004, **109**(3):433-438.
16. St-Onge MP, Janssen I, Heymsfield SB: **Metabolic syndrome in normal-weight Americans new definition of the metabolically obese, normal-weight individual.** Diabetes Care 2004, **27**(9):2222-2228.
17. Liberopoulos E, Mikhailidis D, Elisaf M: **Diagnosis and management of the metabolic syndrome in obesity.** Obesity Reviews 2005, **6**(4):283-296.
18. Finkelstein EA, Khavjou OA, Thompson H, Trogon JG, Pan L, Sherry B, Dietz W: **Obesity and severe obesity forecasts through 2030.** Am J Prev Med 2012, **42**(6):563-570.
19. Ervin RB: **Prevalence of metabolic syndrome among adults 20 years of age and over, by sex, age, race and ethnicity, and body mass index: United States.** National Health Statistics Reports 2009, **13**:1-8.
20. Ford ES, Giles WH, Dietz WH: **Prevalence of the metabolic syndrome among US adults.** JAMA: The Journal of the American Medical Association 2002, **287**(3):356-359.
21. Steinbaum SR: **The metabolic syndrome: an emerging health epidemic in women.** Prog Cardiovasc Dis 2004, **46**(4):321-336.
22. Mokdad AH: **Prevalence of obesity, diabetes, and obesity-related health risk factors, 2001.** JAMA: The Journal of the American Medical Association 2003, **289**(1):76-79.

23. CDC: **Leading causes of death in females United States, 2009** [<http://www.cdc.gov/women/lcod/2009/index.htm>]
24. Farrell SW, Cheng YJ, Blair SN: **Prevalence of the metabolic syndrome across cardiorespiratory fitness levels in women.** *Obes Res* 2012, **12**(5):824-830.
25. Cook S, Weitzman M, Auinger P, Nguyen M, Dietz WH: **Prevalence of a metabolic syndrome phenotype in adolescents: findings from the third National Health and Nutrition Examination Survey, 1988-1994.** *Arch Pediatr Adolesc Med* 2003, **157**(8):821.
26. Larsen D, Murray-Davis M: **A tale of two diets: what can we learn from the diet wars?** *Health Educator* 2005, **37**(1):22-27.
27. Blackburn GL: **Weight of the nation: moving forward, reversing the trend using medical care.** *Am J Clin Nutr* 2012, **96**(5):949-950.
28. Grundy SM, Abate N, Chandalia M: **Diet composition and the metabolic syndrome: what is the optimal fat intake?** *Am J Med* 2002, **113**(9):25-29.
29. Finley CE, LaMonte MJ, Waslien CI, Barlow CE, Blair SN, Nichaman MZ: **Cardiorespiratory fitness, macronutrient intake, and the metabolic syndrome: the Aerobics Center Longitudinal Study.** *J Am Diet Assoc* 2006, **106**(5):673-679.
30. Brunner E, Wunsch H, Marmot M: **What is an optimal diet? Relationship of macronutrient intake to obesity, glucose tolerance, lipoprotein cholesterol levels and the metabolic syndrome in the Whitehall II study.** *International Journal of Obesity and Related Metabolic Disorders: Journal of the International Association for the Study of Obesity* 2001, **25**(1):45.
31. Samaha FF, Iqbal N, Seshadri P, Chicano KL, Daily DA, McGrory J, Williams T, Williams M, Gracely EJ, Stern L: **A low-carbohydrate as compared with a low-fat diet in severe obesity.** *N Engl J Med* 2003, **348**(21):2074-2081.
32. Stern L, Iqbal N, Seshadri P, Chicano KL, Daily DA, McGrory J, Williams M, Gracely EJ, Samaha FF: **The effects of low-carbohydrate versus conventional weight loss diets in severely obese adults: one-year follow-up of a randomized trial.** *Ann Intern Med* 2004, **140**(10):778-785.
33. Stone NJ, Saxon D: **Approach to treatment of the patient with metabolic syndrome: lifestyle therapy.** *American Journal of Cardiology* 2005, **96**(4):15-21.

34. Meckling KA, Gauthier M, Grubb R, Sanford J: **Effects of a hypocaloric, low-carbohydrate diet on weight loss, blood lipids, blood pressure, glucose tolerance, and body composition in free-living overweight women.** Can J Physiol Pharmacol 2002, **80**(11):1095-1105.
35. Westman EC, Yancy WS, Edman JS, Tomlin KF, Perkins CE: **Effect of 6-month adherence to a very low carbohydrate diet program.** Am J Med 2002, **113**(1):30-36.
36. Hickey JT, Hickey L, Yancy Jr WS, Hepburn J, Westman EC: **Clinical use of a carbohydrate-restricted diet to treat the dyslipidemia of the metabolic syndrome.** Metabolic Syndrome and Related Disorders 2003, **1**(3):227-232.
37. Boden G, Sargrad K, Homko C, Mozzoli M, Stein TP: **Effect of a low-carbohydrate diet on appetite, blood glucose levels, and insulin resistance in obese patients with type 2 diabetes.** Ann Intern Med 2005, **142**(6):403-411.
38. Yancy Jr WS, Foy M, Chalecki AM, Vernon MC, Westman EC: **A low-carbohydrate, ketogenic diet to treat type 2 diabetes.** Nutr Metab (Lond) 2005, **2**:34.
39. Hayes MR, Miller CK, Ulbrecht JS, Mauger JL, Parker-Klees L, Gutschall MD, Mitchell DC, Smiciklas-Wright H, Covasa M: **A carbohydrate-restricted diet alters gut peptides and adiposity signals in men and women with metabolic syndrome.** J Nutr 2007, **137**(8):1944-1950.
40. Muzio F, Mondazzi L, Harris WS, Sommariva D, Branchi A: **Effects of moderate variations in the macronutrient content of the diet on cardiovascular disease risk factors in obese patients with the metabolic syndrome.** Am J Clin Nutr 2007, **86**(4):946-951.
41. Piatti P, Monti L, Magni F, Fermo I, Baruffaldi L, Nasser R, Santambrogio G, Librenti M, Galli-Kienle M, Pontiroli A: **Hypocaloric high-protein diet improves glucose oxidation and spares lean body mass: comparison to hypocaloric high-carbohydrate diet.** Metab Clin Exp 1994, **43**(12):1481-1487.
42. Wycherley TP, Noakes M, Clifton PM, Cleanthous X, Keogh JB, Brinkworth GD: **A high-protein diet with resistance exercise training improves weight loss and body composition in overweight and obese patients with type 2 diabetes.** Diabetes Care 2010, **33**(5):969-976.
43. Layman DK, Evans E, Baur JI, Seyler J, Erickson DJ, Boileau RA: **Dietary protein and exercise have additive effects on body composition during weight loss in adult women.** Journal of Nutrition 2005, **135**(8):1903-1910.

44. Kohl H: **Physical activity and cardiovascular disease: evidence for a dose response.** *Medicine & Science in Sport and Exercise* 2001, **33**(6):S472-S483.
45. Rennie KL, McCarthy N, Yazdgerdi S, Marmot M, Brunner E: **Association of the metabolic syndrome with both vigorous and moderate physical activity.** *Int J Epidemiol* 2003, **32**(4):600-606.
46. Bassuk SS, Manson JAE: **Epidemiological evidence for the role of physical activity in reducing risk of type 2 diabetes and cardiovascular disease.** *J Appl Physiol* 2005, **99**(3):1193-1204.
47. Haskell W, Lee I, Pate R, Powell K, Blair S, Franklin B, Macera C, Heath G, Thompson P, Bauman A: **Physical activity and public health: updated recommendation for adults from the American College of Sports Medicine and the American Heart Association.** *Circulation* 2007, **116**(9):1081-1093.
48. Kahn R, Buse J, Ferrannini E, Stern M: **The metabolic syndrome: time for a critical appraisal: joint statement from the American Diabetes Association and the European Association for the Study of Diabetes.** *Diabetes Care* 2005, **28**(9):2289-2304.
49. Lakka TA, Laaksonen DE, Lakka HM, Männikkö N, Niskanen LK, Rauramaa R, Salonen JT: **Sedentary lifestyle, poor cardiorespiratory fitness, and the metabolic syndrome.** *Med Sci Sports Exerc* 2003, **35**(8):1279.
50. Schneider JG, Tompkins C, Blumenthal RS, Mora S: **The metabolic syndrome in women.** *Cardiol Rev* 2006, **14**(6):286-291.
51. Carroll S, Dudfield M: **What is the relationship between exercise and metabolic abnormalities?: A review of the metabolic syndrome.** *Sports Medicine* 2004, **34**(6):371-418.
52. Okura T, Nakata Y, Ohkawara K, Numao S, Katayama Y, Matsuo T, Tanaka K: **Effects of aerobic exercise on metabolic syndrome improvement in response to weight reduction.** *Obesity* 2012, **15**(10):2478-2484.
53. Gaesser G: **Exercise for prevention and treatment of cardiovascular disease, type 2 diabetes, and metabolic syndrome.** *Current Diabetes Reports* 2007, **7**(1):14-19.
54. Swain DP, Franklin BA: **Comparison of cardioprotective benefits of vigorous versus moderate intensity aerobic exercise.** *Am J Cardiol* 2006, **97**(1):141.
55. Westerterp-Plantenga MS, Lemmens SG, Westerterp KR: **Dietary protein—its role in satiety, energetics, weight loss and health.** *Br J Nutr* 2012, **108**(S2):S105-S112.

56. Irwin ML, Ainsworth BE, Mayer-Davis EJ, Addy CL, Pate RR, Durstine JL: **Physical activity and the metabolic syndrome in a tri-ethnic sample of women.** *Obes Res* 2012, **10**(10):1030-1037.
57. Hillier TA, Fagot-Campagna A, Eschwège E, Vol S, Cailleau M, Balkau B: **Weight change and changes in the metabolic syndrome as the French population moves towards overweight: the DESIR cohort.** *Int J Epidemiol* 2006, **35**(1):190-196.
58. Pasanisi F, Contaldo F, De Simone G, Mancini M: **Benefits of sustained moderate weight loss in obesity.** *Nutrition, Metabolism, and Cardiovascular Diseases: NMCD* 2001, **11**(6):401.
59. BMI OC: **Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults.** NOE Initiative; 1998.
60. Grundy SM, Hansen B, Smith Jr SC, Cleeman JI, Kahn RA: **Clinical management of metabolic syndrome.** *Circulation* 2004, **109**(4):551-556.
61. Kerkick C, Thomas A, Campbell B, Taylor L, Wilborn C, Marcello B, Roberts M, Pfau E, Grimstvedt M, Opusunju J, Magrans-Courtney T, Rasmussen C, Wilson R, Kreider RB: **Effects of a popular exercise and weight loss program on weight loss, body composition, energy expenditure and health in obese women.** *Nutrition & Metabolism* 2009, **6**(23):1-17.
62. Kerkick C, Wismann-Bunn J, Fogt D, Thomas A, Taylor L, Campbell B, Wilborn C, Harvey T, Roberts M, Bounty PL, Galbreath M, Marcello B, Rasmussen C, Kreider R: **Changes in weight loss, body composition and cardiovascular disease risk after altering macronutrient distributions during a regular exercise program in obese women.** *Nutrition Journal* 2010, **9**:59.
63. Kreider RB, Rasmussen C, Kerkick CM, Wilborn C, Taylor L, 4th, Campbell B, Magrans-Courtney T, Fogt D, Ferreira M, Li R, Galbreath M, Iosia M, Cooke M, Serra M, Gutierrez J, Byrd M, Kresta JY, Simbo S, Oliver J, Greenwood M: **A carbohydrate-restricted diet during resistance training promotes more favorable changes in body composition and markers of health in obese women with and without insulin resistance.** *Phys Sportsmed* 2011, **39**(2):27-40.
64. Cameron AJ, Shaw JE, Zimmet PZ: **The metabolic syndrome: prevalence in worldwide populations.** *Endocrinol Metab Clin North Am* 2004, **33**(2):351-375.
65. Last JM: *A Dictionary of Epidemiology*: 3rd ed. NY: Oxford University Press; 1995.

66. Sorrentino MJ: **Cholesterol reduction to prevent CAD. What do the data show?** Postgrad Med 2000, **108**(7):40.
67. Campbell DD, Meckling KA: **Effect of the protein: carbohydrate ratio in hypoenergetic diets on metabolic syndrome risk factors in exercising overweight and obese women.** Br J Nutr 2012, **1**(1):1-14.
68. Athyros VG, Mikhailidis DP, Papageorgiou AA, Didangelos TP, Ganotakis ES, Symeonidis AN, Daskalopoulou SS, Kakafika AI, Elisaf M: **Prevalence of atherosclerotic vascular disease among subjects with the metabolic syndrome with or without diabetes mellitus: the METS-GREECE Multicentre Study.** Current Medical Research and Opinion 2004, **20**(11):1691-1701.
69. Adams RJ, Appleton S, Wilson DH, Taylor AW, Dal Grande E, Chittleborough C, Gill T, Ruffin R: **Population comparison of two clinical approaches to the metabolic syndrome implications of the new International Diabetes Federation consensus definition.** Diabetes Care 2005, **28**(11):2777-2779.
70. Athyros VG, Ganotakis ES, Elisaf M, Mikhailidis DP: **The prevalence of the metabolic syndrome using the National Cholesterol Educational Program and International Diabetes Federation definitions.** Current Medical Research and Opinion 2005, **21**(8):1157-1159.
71. Rutter MK, Meigs JB, Sullivan LM, D'Agostino RB, Wilson PW: **Insulin resistance, the metabolic syndrome, and incident cardiovascular events in the Framingham Offspring Study.** Diabetes 2005, **54**(11):3252-3257.
72. ALLHAT O: **Major outcomes in moderately hypercholesterolemic, hypertensive patients randomized to pravastatin vs usual care: the Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial (ALLHAT-LLT).** JAMA: The Journal of the American Medical Association 2002, **288**(23):2998.
73. Gress TW, Nieto FJ, Shahar E, Wofford MR, Brancati FL: **Hypertension and antihypertensive therapy as risk factors for type 2 diabetes mellitus.** N Engl J Med 2000, **342**(13):905-912.
74. Stern MP, Williams K, Hunt KJ: **Impact of diabetes/metabolic syndrome in patients with established cardiovascular disease.** Atherosclerosis Supplements 2005, **6**(2):3.
75. Wang Y, Beydoun MA, Liang L, Caballero B, Kumanyika SK: **Will all Americans become overweight or obese? Estimating the progression and cost of the US obesity epidemic.** Obesity 2008, **16**(10):2323-2330.

76. Abegunde D, Vita-Finzi L: Preventing Chronic Diseases : A Vital Investment. Geneva: World Health Organization; 2005.
77. Bi Y, Wang T, Xu M, Xu Y, Li M, Lu J, Zhu X, Ning G: **Advanced research on risk factors of type 2 diabetes.** Diabetes Metab Res 2012, **28**(s2):32-39.
78. Dwyer JT, Stone EJ, Yang M, Webber LS, Must A, Feldman HA, Nader PR, Perry CL, Parcel GS: **Prevalence of marked overweight and obesity in a multiethnic pediatric population: findings from the Child and Adolescent Trial for Cardiovascular Health (CATCH) study.** J Am Diet Assoc 2000, **100**(10):1149-1154.
79. Lawson M, Chen L, Daniels S, Dolan L: **Prevalance of morbid obesity and metabolic syndrome in U.S. adolescents and very young adults.** Ann Epidemiol 2005, **15**(8):639.
80. Lundberg V: **Diabetes as a risk factor for myocardial infarction: population and gender perspectives.** Journal of Internal Medicine 1997, **241**(6):485-492.
81. Ramos RG: **The prevalence of metabolic syndrome among US women of childbearing age.** American Journal of Public Health 2008, **98**(6):1122-1127.
82. Rosamond W, Flegal K, Friday G, et al.: **Heart disease and stroke statistics - 2007 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee.** Circulation 2007, **115**(4):e69-e171.
83. Kerksick C, Thomas A, Campbell B, Taylor L, Wilborn C, Marcello B, Roberts M, Pfau E, Grimstvedt M, Opusunju J, Magrans-Courtney T, Rasmussen C, Wilson R, Kreider RB: **Effects of a popular exercise and weight loss program on weight loss, body composition, energy expenditure and health in obese women.** Nutrition & Metabolism 2009, **1**:1-17; x.
84. Ornish D, Scherwitz LW, Billings JH, Gould KL, Merritt TA, Sparler S, Armstrong WT, Ports TA, Kirkeeide RL, Hogeboom C: **Intensive lifestyle changes for reversal of coronary heart disease.** JAMA: The Journal of the American Medical Association 1998, **280**(23):2001-2007.
85. Cranford LS: **Diagnosis, prevention, and intervention for the metabolic syndrome.** The American Journal of Cardiology 2003, **92**(1, Supplement 1):35-42.
86. Knopp RH, Walden CE, Retzlaff BM, McCann BS, Dowdy AA, Albers JJ, Gey GO, Cooper MN: **Long-term cholesterol-lowering effects of 4 fat-restricted diets in hypercholesterolemic and combined hyperlipidemic men.** JAMA: The Journal of the American Medical Association 1997, **278**(18):1509-1515.

87. Carnethon MR, Loria CM, Hill JO, Sidney S, Savage PJ, Liu K: **Risk factors for the metabolic syndrome - the Coronary Artery Risk Development in Young Adults (CARDIA) study, 1985–2001.** Diabetes Care 2004, **27**(11):2707-2715.
88. Merchant AT, Vatanparast H, Barlas S, Dehghan M, Shah SMA, De Koning L, Steck SE: **Carbohydrate intake and overweight and obesity among healthy adults.** J Am Diet Assoc 2009, **109**(7):1165.
89. Miller CK, Ulbrecht JS, Lyons J, Parker-Klees L, Gutschall MD, Smiciklas-Wright H, Mitchell DC, Covasa M, Hayes M: **A reduced-carbohydrate diet improves outcomes in patients with metabolic syndrome: a translational study.** Topics in Clinical Nutrition 2007, **22**(1):82.
90. Sasakabe T, Haimoto H, Umegaki H, Wakai K: **Effects of a moderate low-carbohydrate diet on preferential abdominal fat loss and cardiovascular risk factors in patients with type 2 diabetes.** Diabetes, Metabolic Syndrome and Obesity: Targets and Therapy 2011, **4**:167.
91. Saris W, Astrup A, Prentice A, Zunft H, Formiguera X, Verboeket-van de Venne W, Raben A, Poppitt S, Seppelt B, Johnston S: **Randomized controlled trial of changes in dietary carbohydrate/fat ratio and simple vs complex carbohydrates on body weight and blood lipids: the CARMEN study.** Int J Obes 2000, **24**(10):1310-1318.
92. Poppitt SD, Keogh GF, Prentice AM, Williams DE, Sonnemans HM, Valk EE, Robinson E, Wareham NJ: **Long-term effects of ad libitum low-fat, high-carbohydrate diets on body weight and serum lipids in overweight subjects with metabolic syndrome.** Am J Clin Nutr 2002, **75**(1):11-20.
93. Tuomilehto J, Lindström J, Eriksson JG, Valle TT, Hämäläinen H, Ilanne-Parikka P, Keinänen-Kiukaanniemi S, Laakso M, Louheranta A, Rastas M: **Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance.** N Engl J Med 2001, **344**(18):1343-1350.
94. Goldstein T, Kark JD, Berry EM, Adler B, Ziv E, Raz I: **The effect of a low carbohydrate energy-unrestricted diet on weight loss in obese type 2 diabetes patients - a randomized controlled trial.** e-SPEN, The European e-Journal of Clinical Nutrition and Metabolism 2011, **6**(4):e178-e186.
95. Hussain TA, Mathew TC, Dashti AA, Asfar S, Al-Zaid N, Dashti HM: **Effect of low-calorie versus low-carbohydrate ketogenic diet in type 2 diabetes.** Nutrition 2012, **28**(10):1016-1021.

96. Rodríguez-Villar C, Manzanares JM, Casals E, Pérez-Heras A, Zambón D, Gomis R, Ros E: **High-monounsaturated fat, olive oil-rich diet has effects similar to a high-carbohydrate diet on fasting and postprandial state and metabolic profiles of patients with type 2 diabetes.** *Metab Clin Exp* 2000, **49**(12):1511-1517.
97. Noakes M, Clifton PM: **Changes in plasma lipids and other cardiovascular risk factors during 3 energy-restricted diets differing in total fat and fatty acid composition.** *Am J Clin Nutr* 2000, **71**(3):706-712.
98. Landry N, Bergeron N, Archer R, Samson P, Corneau L, Bergeron J, Dériaz O: **Whole-body fat oxidation rate and plasma triacylglycerol concentrations in men consuming an ad libitum high-carbohydrate or low-carbohydrate diet.** *Am J Clin Nutr* 2003, **77**(3):580-586.
99. Colette C, Percheron C, Pares-Herbute N, Michel F, Pham T, Brillant L, Descomps B, Monnier L: **Exchanging carbohydrates for monounsaturated fats in energy-restricted diets: effects on metabolic profile and other cardiovascular risk factors.** *Int J Obes* 2003, **27**(6):648-656.
100. Brehm BJ, Seeley RJ, Daniels SR, D'Alessio DA: **A randomized trial comparing a very low carbohydrate diet and a calorie-restricted low fat diet on body weight and cardiovascular risk factors in healthy women.** *Journal of Clinical Endocrinology & Metabolism*; 2003 2003, **88**(4):1617-1623.
101. Volek JS, Sharman MJ, Gómez AL, DiPasquale C, Roti M, Pumerantz A, Kraemer WJ: **Comparison of a very low-carbohydrate and low-fat diet on fasting lipids, LDL subclasses, insulin resistance, and postprandial lipemic responses in overweight women.** *J Am Coll Nutr* 2004, **23**(2):177-184.
102. Miyashita Y, Koide N, Ohtsuka M, Ozaki H, Itoh Y, Oyama T, Uetake T, Ariga K, Shirai K: **Beneficial effect of low carbohydrate in low calorie diets on visceral fat reduction in type 2 diabetic patients with obesity.** *Diabetes Res Clin Pract* 2004, **65**(3):235-242.
103. Gerhard GT, Ahmann A, Meeuws K, McMurry MP, Duell PB, Connor WE: **Effects of a low-fat diet compared with those of a high-monounsaturated fat diet on body weight, plasma lipids and lipoproteins, and glycemic control in type 2 diabetes.** *Am J Clin Nutr* 2004, **80**(3):668-673.
104. Meckling KA, O'Sullivan C, Saari D: **Comparison of a low-fat diet to a low-carbohydrate diet on weight loss, body composition, and risk factors for diabetes and cardiovascular disease in free-living, overweight men and women.** *Journal of Clinical Endocrinology & Metabolism* 2004, **89**(6):2717-2723.

105. Yancy WS, Olsen MK, Guyton JR, Bakst RP, Westman EC: **A low-carbohydrate, ketogenic diet versus a low-fat diet to treat obesity and hyperlipidemia - a randomized, controlled trial.** Ann Intern Med 2004, **140**(10):769-777.
106. Shai I, Schwarzfuchs D, Henkin Y, Shahar DR, Witkow S, Greenberg I, Golan R, Fraser D, Bolotin A, Vardi H: **Weight loss with a low-carbohydrate, Mediterranean, or low-fat diet.** N Engl J Med 2008, **359**(3):229-241.
107. Davis NJ, Tomuta N, Schechter C, Isasi CR, Segal-Isaacson C, Stein D, Zonszein J, Wylie-Rosett J: **Comparative study of the effects of a 1-year dietary intervention of a low-carbohydrate diet versus a low-fat diet on weight and glycemic control in type 2 diabetes.** Diabetes Care 2009, **32**(7):1147-1152.
108. Brehm BJ, Lattin BL, Summer SS, Boback JA, Gilchrist GM, Jandacek RJ, D'Alessio DA: **One-year comparison of a high-monounsaturated fat diet with a high-carbohydrate diet in type 2 diabetes.** Diabetes Care 2009, **32**(2):215-220.
109. Mueller C, Masri B, Hogg J, Mastrogiacono M, Chiu Y: **Carbohydrate- vs fat-controlled diet effect on weight loss and coronary artery disease risk. A pilot feeding study.** Nutrition in Clinical Practice 2010, **25**(5):542-547.
110. Foster GD, Wyatt HR, Hill JO, Makris AP, Rosenbaum DL, Brill C, Stein RI, Mohammed BS, Miller B, Rader DJ: **Weight and metabolic outcomes after 2 years on a low-carbohydrate versus low-fat diet: a randomized trial.** Ann Intern Med 2010, **153**(3):147.
111. Rajaie Somayeh Al, Khazaei M, Esmailzadeh A: **Effects of moderately-restricted carbohydrate diet on cardiovascular risk factors among women with metabolic syndrome.** Journal of Ishahan Medical School (I.U.M.S) **29**(171):0.
112. Metkus TS, Dobrosielski D, Stewart K: **Effect of a low carbohydrate versus a low fat diet on the metabolic syndrome.** J Am Coll Cardiol 2012, **59**(13s1):E1753-E1753.
113. Iqbal N, Vetter ML, Moore RH, Chittams JL, Dalton-Bakes CV, Dowd M, Williams-Smith C, Cardillo S, Wadden TA: **Effects of a low-intensity intervention that prescribed a low-carbohydrate vs. a low-fat diet in obese, diabetic participants.** Obesity 2009, **18**(9):1733-1738.
114. National Research Council (US). Subcommittee on the Tenth Edition of the RDAs: Recommended dietary allowances. National Academies Press; 1989.

115. Eisenstein J, Roberts SB, Dallal G, Saltzman E: **High-protein weight-loss diets: are they safe and do they work? A review of the experimental and epidemiologic data.** Nutr Rev 2008, **60**(7):189-200.
116. Smit E, Nieto FJ, Crespo CJ, Mitchell P: **Estimates of animal and plant protein intake in US adults: results from the Third National Health and Nutrition Examination Survey, 1988-1991.** J Am Diet Assoc 1999, **99**(7):813-820.
117. Institute of Medicine (US). Panel on Macronutrients, Institute of Medicine (US). Standing Committee on the Scientific Evaluation of Dietary Reference Intakes: Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids. National Academy Press; 2005.
118. Wolfe RR: **The underappreciated role of muscle in health and disease.** American Journal of Clinical Nutrition 2006, **84**(3):475-482.
119. Krieger JW, Sitren HS, Daniels MJ, Langkamp-Henken B: **Effects of variation in protein and carbohydrate intake on body mass and composition during energy restriction: a meta-regression.** Am J Clin Nutr 2006, **83**(2):260-274.
120. Meckling K, Sherfey R: **A randomized trial of a hypocaloric high-protein diet, with and without exercise, on weight loss, fitness, and markers of the metabolic syndrome in overweight and obese women.** Applied Physiology, Nutrition, and Metabolism 2007, **32**(4):743-752.
121. Kreider R, Culbertson J, Byrd M, Simbo S, Oliver J, Mardock M, Cannon C, Yung Y, Khanna D, Koozehchian M: **Maintaining a high protein diet while participating in a resistance training program does not affect markers of bone health in women.** The Journal of Strength & Conditioning Research 2011, **25**:S71.
122. Layman DK, Boileau RA, Erickson DJ, Painter JE, Shiue H, Sather C, Christou DD: **A reduced ratio of dietary carbohydrate to protein improves body composition and blood lipid profiles during weight loss in adult women.** Journal of Nutrition 2003, **133**(2):411-417.
123. Volek JS, Sharman MJ, Gómez AL, Scheett TP, Kraemer WJ: **An isoenergetic very low carbohydrate diet improves serum HDL cholesterol and triacylglycerol concentrations, the total cholesterol to HDL cholesterol ratio and postprandial lipemic responses compared with a low fat diet in normal weight, normolipidemic women.** J Nutr 2003, **133**(9):2756-2761.

124. Gannon MC, Nuttall FQ, Saeed A, Jordan K, Hoover H: **An increase in dietary protein improves the blood glucose response in persons with type 2 diabetes.** Am J Clin Nutr 2003, **78**(4):734-741.
125. Farnsworth E, Luscombe ND, Noakes M, Wittert G, Argyiou E, Clifton PM: **Effect of a high-protein, energy-restricted diet on body composition, glycemic control, and lipid concentrations in overweight and obese hyperinsulinemic men and women.** Am J Clin Nutr 2003, **78**(1):31-39.
126. Foster GD, Wyatt HR, Hill JO, McGuckin BG, Brill C, Mohammed BS, Szapary PO, Rader DJ, Edman JS, Klein S: **A randomized trial of a low-carbohydrate diet for obesity.** N Engl J Med 2003, **348**(21):2082-2090.
127. Gannon MC, Nuttall FQ: **Effect of a high-protein, low-carbohydrate diet on blood glucose control in people with type 2 diabetes.** Diabetes 2004, **53**(9):2375-2382.
128. Sharman MJ, Gomez AL, Kraemer WJ, Volek JS: **Very low-carbohydrate and low-fat diets affect fasting lipids and postprandial lipemia differently in overweight men.** J Nutr 2004, **134**(4):880-885.
129. Appel LJ, Sacks FM, Carey VJ, Obarzanek E, Swain JF, Miller III ER, Conlin PR, Erlinger TP, Rosner BA, Laranjo NM: **Effects of protein, monounsaturated fat, and carbohydrate intake on blood pressure and serum lipids.** JAMA: The Journal of the American Medical Association 2005, **294**(19):2455-2464.
130. McAuley K, Smith K, Taylor R, McLay R, Williams S, Mann J: **Long-term effects of popular dietary approaches on weight loss and features of insulin resistance.** Int J Obes 2005, **30**(2):342-349.
131. Keogh JB, Brinkworth GD, Noakes M, Belobrajdic DP, Buckley JD, Clifton PM: **Effects of weight loss from a very-low-carbohydrate diet on endothelial function and markers of cardiovascular disease risk in subjects with abdominal obesity.** Am J Clin Nutr 2008, **87**(3):567-576.
132. Tay J, Brinkworth GD, Noakes M, Keogh J, Clifton PM: **Metabolic effects of weight loss on a very-low-carbohydrate diet compared with an isocaloric high-carbohydrate diet in abdominally obese subjects.** J Am Coll Cardiol 2008, **51**(1):59-67.
133. Jenkins DJ, Wong JM, Kendall CW, Esfahani A, Ng VW, Leong TC, Faulkner DA, Vidgen E, Greaves KA, Paul G: **The effect of a plant-based low-carbohydrate ("Eco-Atkins") diet on body weight and blood lipid concentrations in hyperlipidemic subjects.** Arch Intern Med 2009, **169**(11):1046.

134. Volek JS, Phinney SD, Forsythe CE, Quann EE, Wood RJ, Puglisi MJ, Kraemer WJ, Bibus DM, Fernandez ML, Feinman RD: **Carbohydrate restriction has a more favorable impact on the metabolic syndrome than a low fat diet.** *Lipids* 2009, **44**(4):297-309.
135. Claessens M, Van Baak M, Monsheimer S, Saris W: **The effect of a low-fat, high-protein or high-carbohydrate ad libitum diet on weight loss maintenance and metabolic risk factors.** *Int J Obes* 2009, **33**(3):296-304.
136. Brinkworth GD, Noakes M, Buckley JD, Keogh JB, Clifton PM: **Long-term effects of a very-low-carbohydrate weight loss diet compared with an isocaloric low-fat diet after 12 mo.** *Am J Clin Nutr* 2009, **90**(1):23-32.
137. Lim SS, Noakes M, Keogh JB, Clifton PM: **Long-term effects of a low carbohydrate, low fat or high unsaturated fat diet compared to a no-intervention control.** *Nutrition, Metabolism and Cardiovascular Diseases* 2010, **20**(8):599-607.
138. Papakonstantinou E, Triantafyllidou D, Panagiotakos D, Koutsovasilis A, Saliaris M, Manolis A, Melidonis A, Zampelas A: **A high-protein low-fat diet is more effective in improving blood pressure and triglycerides in calorie-restricted obese individuals with newly diagnosed type 2 diabetes.** *Eur J Clin Nutr* 2010, **64**(6):595-602.
139. Flechtner-Mors M, Boehm BO, Wittmann R, Thoma U, Ditschuneit HH: **Enhanced weight loss with protein-enriched meal replacements in subjects with the metabolic syndrome.** *Diabetes Metab Res* 2010, **26**(5):393-405.
140. Sargrad KR, Homko C, Mozzoli M, Boden G: **Effect of high protein vs high carbohydrate intake on insulin sensitivity, body weight, hemoglobin A1c, and blood pressure in patients with type 2 diabetes mellitus.** *J Am Diet Assoc* 2005, **105**(4):573-580.
141. Hodgson JM, Burke V, Beilin LJ, Puddey IB: **Partial substitution of carbohydrate intake with protein intake from lean red meat lowers blood pressure in hypertensive persons.** *Am J Clin Nutr* 2006, **83**(4):780-787.
142. Te Morenga LA, Levers MT, Williams SM, Brown RC, Mann J: **Comparison of high protein and high fiber weight-loss diets in women with risk factors for the metabolic syndrome: a randomized trial.** *Nutr J* 2011, **10**(40):40.
143. Larsen R, Mann N, Maclean E, Shaw J: **The effect of high-protein, low-carbohydrate diets in the treatment of type 2 diabetes: a 12 month randomised controlled trial.** *Diabetologia* 2011, **54**(4):731-740.

144. Due A, Toubro S, Skov A, Astrup A: **Effect of normal-fat diets, either medium or high in protein, on body weight in overweight subjects: a randomised 1-year trial.** *Int J Obes* 2004, **28**(10):1283-1290.
145. Te Morenga L, Williams S, Brown R, Mann J: **Effect of a relatively high-protein, high-fiber diet on body composition and metabolic risk factors in overweight women.** *Eur J Clin Nutr* 2010, **64**(11):1323-1331.
146. Parker B, Noakes M, Luscombe N, Clifton P: **Effect of a high-protein, high-monounsaturated fat weight loss diet on glycemic control and lipid levels in type 2 diabetes.** *Diabetes Care* 2002, **25**(3):425-430.
147. Lee K, Lee J, Bae W, Choi J, Kim H, Cho B: **Efficacy of low-calorie, partial meal replacement diet plans on weight and abdominal fat in obese subjects with metabolic syndrome: a double-blind, randomised controlled trial of two diet plans—one high in protein and one nutritionally balanced.** *Int J Clin Pract* 2009, **63**(2):195-201.
148. Lopez-Jimenez F, Xu L, Edens KL: **Improvement of metabolic syndrome with low refined carbohydrates, relatively high protein diet enriched with mono- and poly-unsaturated fatty acids.** *J Am Coll Cardiol* 2010, **55**(10s1):A58. E550.
149. Johnston CS, Tjonn SL, Swan PD: **High-protein, low-fat diets are effective for weight loss and favorably alter biomarkers in healthy adults.** *J Nutr* 2004, **134**(3):586-591.
150. Brinkworth GD, Noakes M, Parker B, Foster P, Clifton PM: **Long-term effects of advice to consume a high-protein, low-fat diet, rather than a conventional weight-loss diet, in obese adults with type 2 diabetes: one-year follow-up of a randomised trial.** *Diabetologia* 2004, **47**(10):1677-1686.
151. Brinkworth GD, Noakes M, Keogh JB, Luscombe N, Wittert GA, Clifton PM: **Long-term effects of a high-protein, low-carbohydrate diet on weight control and cardiovascular risk markers in obese hyperinsulinemic subjects.** *Int J Obes* 2004, **28**(5):661-670.
152. Luscombe-Marsh ND, Noakes M, Wittert GA, Keogh JB, Foster P, Clifton PM: **Carbohydrate-restricted diets high in either monounsaturated fat or protein are equally effective at promoting fat loss and improving blood lipids.** *Am J Clin Nutr* 2005, **81**(4):762-772.

153. Leidy HJ, Carnell NS, Mattes RD, Campbell WW: **Higher protein intake preserves lean mass and satiety with weight loss in pre-obese and obese women.** Obesity 2007, **15**(2):421-429.
154. Clifton PM, Keogh JB, Noakes M: **Long-term effects of a high-protein weight-loss diet.** Am J Clin Nutr 2008, **87**(1):23-29.
155. Toscani MK, Mario FM, Radavelli-Bagatini S, Wiltgen D, Cristina Matos M, Spritzer PM: **Effect of high-protein or normal-protein diet on weight loss, body composition, hormone, and metabolic profile in southern Brazilian women with polycystic ovary syndrome: a randomized study.** Gynecological Endocrinology 2011, **27**(11):925-930.
156. Pearce KL, Clifton PM, Noakes M: **Egg consumption as part of an energy-restricted high-protein diet improves blood lipid and blood glucose profiles in individuals with type 2 diabetes.** Br J Nutr 2011, **105**(4):584.
157. Mokdad AH, Marks JS, Stroup DF, Gerberding JL: **Actual causes of death in the United States, 2000.** JAMA: The Journal of the American Medical Association 2004, **291**(10):1238-1245.
158. Lavie CJ, Milani RV: **Cardiac rehabilitation and exercise training programs in metabolic syndrome and diabetes.** Journal of Cardiopulmonary Rehabilitation and Prevention 2005, **25**(2):59-66.
159. Hu FB, Li TY, Colditz GA, Willett WC, Manson JAE: **Television watching and other sedentary behaviors in relation to risk of obesity and type 2 diabetes mellitus in women.** JAMA: The Journal of the American Medical Association 2003, **289**(14):1785-1791.
160. Wessel TR, Arant CB, Olson MB, Johnson BD, Reis SE, Sharaf BL, Shaw LJ, Handberg E, Sopko G, Kelsey SF: **Relationship of physical fitness vs body mass index with coronary artery disease and cardiovascular events in women.** JAMA: The Journal of the American Medical Association 2004, **292**(10):1179-1187.
161. Mensink G, Ziese T, Kok FJ: **Benefits of leisure-time physical activity on the cardiovascular risk profile at older age.** Int J Epidemiol 1999, **28**(4):659-666.
162. Wareham NJ, Hennings SJ, Byrne CD, Hales CN, Prentice AM, Day NE: **A quantitative analysis of the relationship between habitual energy expenditure, fitness and the metabolic cardiovascular syndrome.** Br J Nutr 1998, **80**:235-241.

163. Franks PW, Ekelund U, Brage S, Wong MY, Wareham NJ: **Does the association of habitual physical activity with the metabolic syndrome differ by level of cardiorespiratory fitness?** Diabetes Care 2004, **27**(5):1187-1193.
164. Ekelund U, Brage S, Franks PW, Hennings S, Emms S, Wareham NJ: **Physical activity energy expenditure predicts progression toward the metabolic syndrome independently of aerobic fitness in middle-aged healthy Caucasians The Medical Research Council Ely Study.** Diabetes Care 2005, **28**(5):1195-1200.
165. LaMonte MJ, Barlow CE, Jurca R, Kampert JB, Church TS, Blair SN: **Cardiorespiratory fitness is inversely associated with the incidence of metabolic syndrome - a prospective study of men and women.** Circulation 2005, **112**(4):505-512.
166. Rennie KL, McCarthy N, Yazdgerdi S, Marmot M, Brunner E: **Association of the metabolic syndrome with both vigorous and moderate physical activity.** Int J Epidemiol 2003, **32**(4):600-606.
167. Laaksonen DE, Lakka HM, Salonen JT, Niskanen LK, Rauramaa R, Lakka TA: **Low levels of leisure-time physical activity and cardiorespiratory fitness predict development of the metabolic syndrome.** Diabetes Care 2002, **25**(9):1612-1618.
168. Thompson PD, Crouse SF, Goodpaster B, Kelley D, Moyna N, Pescatello L: **The acute versus the chronic response to exercise.** Med Sci Sports Exerc 2001, **33**(6; SUPP):438-445.
169. Jurca R: **Association of muscular strength with incidence of metabolic syndrome in men.** Medicine and Science in Sports and Exercise 2005, **37**(11):1849-1855.
170. Strasser B: **Resistance training in the treatment of the metabolic syndrome.** Sports Medicine 2010, **40**(5):397-415.
171. Willis LH, Slentz CA, Bateman LA, Shields AT, Piner LW, Bales CW, Houmard JA, Kraus WE: **Effects of aerobic and/or resistance training on body mass and fat mass in overweight or obese adults.** J Appl Physiol 2012, **113**(12):1831-1837.
172. Lee DT: **Effects of a high protein diet and aerobic exercise on body weight changes and blood lipids in slightly overweight women.** Nutritional Sciences 2005, **8**(1):28-34.

173. Noakes M, Keogh JB, Foster PR, Clifton PM: **Effect of an energy-restricted, high-protein, low-fat diet relative to a conventional high-carbohydrate, low-fat diet on weight loss, body composition, nutritional status, and markers of cardiovascular health in obese women.** *Am J Clin Nutr* 2005, **81**(6):1298-1306.
174. McAuley K, Hopkins C, Smith K, McLay R, Williams S, Taylor R, Mann J: **Comparison of high-fat and high-protein diets with a high-carbohydrate diet in insulin-resistant obese women.** *Diabetologia* 2005, **48**(1):8-16.
175. Dansinger ML, Gleason JA, Griffith JL, Selker HP, Schaefer EJ: **Comparison of the Atkins, Ornish, Weight Watchers, and Zone diets for weight loss and heart disease risk reduction.** *JAMA: The Journal of the American Medical Association* 2005, **293**(1):43-53.
176. Ferrara L, Innelli P, Palmieri V, Limauro S, De Luca G, Ferrara F, Liccardo E, Celentano A: **Effects of different dietary protein intakes on body composition and vascular reactivity.** *Eur J Clin Nutr* 2005, **60**(5):643-649.
177. Bowden RG, Lanning BA, Doyle EI, Slonaker B, Johnston HM, Scanes G: **Effects of a high protein diet and exercise on lipid levels.** *Research Quarterly for Exercise and Sport* 2007, **78**(1):A25.
178. Gardner CD, Kiazand A, Alhassan S, Kim S, Stafford RS, Balise RR, Kraemer HC, King AC: **Comparison of the Atkins, Zone, Ornish, and LEARN diets for change in weight and related risk factors among overweight premenopausal women.** *JAMA: The Journal of the American Medical Association* 2007, **297**(9):969-977.
179. Lasker D, Evans EM, Layman DK: **Moderate carbohydrate, moderate protein weight loss diet reduces cardiovascular disease risk compared to high carbohydrate, low protein diet in obese adults: A randomized clinical trial.** *Nutr Metab (Lond)* 2008, **5**:30.
180. Layman DK, Evans EM, Erickson D, Seyler J, Weber J, Bagshaw D, Griel A, Psota T, Kris-Etherton P: **A moderate-protein diet produces sustained weight loss and long-term changes in body composition and blood lipids in obese adults.** *J Nutr* 2009, **139**(3):514-521.
181. Sacks FM, Bray GA, Carey VJ, Smith SR, Ryan DH, Anton SD, McManus K, Champagne CM, Bishop LM, Laranjo N: **Comparison of weight-loss diets with different compositions of fat, protein, and carbohydrates.** *N Engl J Med* 2009, **360**(9):859-873.

182. Josse AR, Atkinson SA, Tarnopolsky MA, Phillips SM: **Increased consumption of dairy foods and protein during diet-and exercise-induced weight loss promotes fat mass loss and lean mass gain in overweight and obese premenopausal women.** J Nutr 2011, **141**(9):1626-1634.
183. Dutheil F, Lac G, Courteix D, Doré E, Chapier R, Roszyk L, Sapin V, Lesourd B: **Treatment of metabolic syndrome by combination of physical activity and diet needs an optimal protein intake: a randomized controlled trial.** Nutrition Journal 2012, **11**(1):72.
184. Expert Panel on Detection/Evaluation T: **Executive summary of the third report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III).** Journal-American Medical Association 2001, **285**(19):2486-2497.
185. Ross R, Dagnone D, Jones PJH, Smith H, Paddags A, Hudson R, Janssen I: **Reduction in obesity and related comorbid conditions after diet-induced weight loss or exercise-induced weight loss in men.** Ann Intern Med 2000, **133**:92-103.
186. De Koning L, Merchant AT, Pogue J, Anand SS: **Waist circumference and waist-to-hip ratio as predictors of cardiovascular events: meta-regression analysis of prospective studies.** Eur Heart J 2007, **28**(7):850-856.
187. Janssen I, Katzmarzyk PT, Ross R: **Waist circumference and not body mass index explains obesity-related health risk.** Am J Clin Nutr 2004, **79**(3):379-384.
188. Willett WC: **Is dietary fat a major determinant of body fat?** Am J Clin Nutr 1998, **67**(3):556S-562S.
189. Buchholz AC, Schoeller DA: **Is a calorie a calorie?** Am J Clin Nutr 2004, **79**(5):899S-906S.
190. Signs CDCV: **Prevalence, treatment, and control of hypertension—United States, 1999–2002 and 2005–2008.** MMWR 2011, **60**:103-108.
191. Roger VL, Go AS, Lloyd-Jones DM, Benjamin EJ, Berry JD, Borden WB, Bravata DM, Dai S, Ford ES, Fox CS: **Heart disease and stroke statistics—2012 update a report from the American Heart Association.** Circulation 2012, **125**(1):e2-e220.
192. Heron M, Hoyert DL, Murphy SL, Xu J: **National vital statistics reports.** National Vital Statistics Reports 2009, **57**(14).
193. Wilson PWF, Grundy SM: **The metabolic syndrome - a practical guide to origins and treatment: part II.** Circulation 2003, **108**(13):1537-1540.

194. Weinsier RL, James L, Darnell B, Wooldridge N, Birch R, Hunter GR, Bartolucci AA: **Lipid and insulin concentrations in obese postmenopausal women: separate effects of energy restriction and weight loss.** Am J Clin Nutr 1992, **56**(1):44-49.
195. Ramsay L, Ramsay M, Hettiarachchi J, Davies D, Winchester J: **Weight reduction in a blood pressure clinic.** Br Med J 1978, **2**(6132):244-245.
196. Collaboration PS: **Age-specific relevance of usual blood pressure to vascular mortality: a meta-analysis of individual data for one million adults in 61 prospective studies.** Lancet (London, England) 2002, **360**(9349):1903-1913.
197. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo Jr JL, Jones DW, Materson BJ, Oparil S, Wright Jr JT: **Seventh report of the joint national committee on prevention, detection, evaluation, and treatment of high blood pressure.** Hypertension 2003, **42**(6):1206-1252.
198. Stevenson RW, McPherson RK, Persson LM, Genereux PE, Swick AG, Spitzer J, Herbst JJ, Andrews KM, Kreutter DK, Gibbs EM: **The antihyperglycemic agent englitazone prevents the defect in glucose transport in rats fed a high-fat diet.** Diabetes 1996, **45**(1):60-6.
199. Hagberg J: Exercise, fitness, and hypertension. Exercise, Fitness, and Health: A consensus of Current Knowledge. Ed: EL Schneider, JW Rowe. Champaign, IL: Human Kinetics Publishers 1990, 455-566.
200. Centers for Disease Control and Prevention (CDC): **Prevalence of diabetes and impaired fasting glucose in adults--United States, 1999-2000.** MMWR Morb Mortal Wkly Rep 2003, **52**(35):833-837.
201. Reaven GM: **Role of insulin resistance in human disease.** Diabetes 1988, **37**(12):1595-1607.
202. Boden G, Chen X, Ruiz J, White J, Rossetti L: **Mechanisms of fatty acid-induced inhibition of glucose uptake.** J Clin Invest 1994, **93**(6):2438.
203. Parker B, Noakes M, Luscombe N, Clifton P: **Effect of a high-protein, high-monounsaturated fat weight loss diet on glycemic control and lipid levels in type 2 diabetes.** Diabetes Care 2002, **25**(3):425-430.
204. Reaven G: **Syndrome X.** Current Treatment Options in Cardiovascular Medicine 2001, **3**(4):323-332.

205. Expert Panel on Detection Evaluation, T.H.B.C.A.: **Executive summary of the third report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III)**. Journal-American Medical Association 2001, **285**(19):2486-2497.
206. Dela F, Mikines KJ, von Linstow M, Secher NH, Galbo H: **Effect of training on insulin-mediated glucose uptake in human muscle**. American Journal of Physiology-Endocrinology And Metabolism 2006, **263**(6):E1134-E1143.
207. Kahn BB: **Glucose transport: pivotal step in insulin action**. Diabetes 1996, **45**(11):1644-1654.
208. Brandenburg SL, Reusch J, Bauer TA, Jeffers BW, Hiatt WR, Regensteiner JG: **Effects of exercise training on oxygen uptake kinetic responses in women with type 2 diabetes**. Diabetes Care 1999, **22**(10):1640-1646.
209. Seo T, Blaner WS, Deckelbaum RJ: **Omega-3 fatty acids: molecular approaches to optimal biological outcomes**. Curr Opin Lipidol 2005, **16**(1):11-18.
210. Knowler WC, Barrett-Connor E, Fowler SE, Hamman RF, Lachin JM, Walker EA, Nathan DM, Diabetes Prevention Program Research Group: **Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin**. N Engl J Med 2002, **346**(6):393-403.
211. Pan XR, Li G, Hu YH, Wang JX, Yang WY, An ZX, Hu ZX, Xiao JZ, Cao HB, Liu PA: **Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance: the Da Qing IGT and Diabetes Study**. Diabetes Care 1997, **20**(4):537-544.
212. Marshall J, Bessesen D, Hamman R: **High saturated fat and low starch and fibre are associated with hyperinsulinaemia in a non-diabetic population: the San Luis Valley Diabetes Study**. Diabetologia 1997, **40**(4):430-438.
213. Eriksson J, Taimela S, Eriksson K, Parviainen S, Peltonen J, Kujala U: **Resistance training in the treatment of non-insulin-dependent diabetes mellitus**. Int J Sports Med 1997, **18**(4):242.
214. Masuzaki H, Paterson J, Shinyama H, Morton NM, Mullins JJ, Seckl JR, Flier JS: **A transgenic model of visceral obesity and the metabolic syndrome**. Science 2001, **294**(5549):2166-2170.
215. Jenkins D, Kendall C, Augustin L, Vuksan V: **High-complex carbohydrate or lente carbohydrate foods?** Am J Med 2002, **113**:30S.

216. Nestel PJ, Whyte HM, Goodman DWS: **Distribution and turnover of cholesterol in humans.** J Clin Invest 1969, **48**(6):982.

217. Couillard C, Després JP, Lamarche B, Bergeron J, Gagnon J, Leon AS, Rao D, Skinner JS, Wilmore JH, Bouchard C: **Effects of endurance exercise training on plasma HDL cholesterol levels depend on levels of triglycerides evidence from men of the Health, Risk Factors, Exercise Training and Genetics (HERITAGE) Family Study.** Arterioscler Thromb Vasc Biol 2001, **21**(7):1226-1232.

218. Heavin GC: *Curves: Permanent Results Without Permanent Dieting.* New York: G.P. Putman's Sons; 2003.

219. Kerkick C, Wismann-Bunn J, Fogt D, Thomas A, Taylor L, Campbell B, Wilborn C, Harvey T, Roberts M, Bounty PL, Galbreath M, Marcello B, Rasmussen C, Kreider R: **Changes in weight loss, body composition and cardiovascular disease risk after altering macronutrient distributions during a regular exercise program in obese women.** Nutrition Journal 2010, **9**:59.

220. ACSM: *ACSM's Guidelines for Exercise Testing and Prescription: 7th ed.* Philadelphia, PA: Lippincott William & Wilkins; 2006.

221. Matarese LE: **Indirect calorimetry: Technical aspects.** Journal of the American Dietetic Association 1997, **97**(10):S154-S160.

222. Glickman SG: **Validity and reliability of dual-energy x-ray absorptiometry for the assessment of abdominal adiposity.** J Appl Physiol 2004, **97**(2):509-514.

223. Kohrt WM: **Preliminary evidence that DEXA provides an accurate assessment of body composition.** J Appl Physiol 1998, **84**(1):372-377.

224. Almada AL: **Comparison of the reliability of repeated whole body DEXA scans to repeated spine and hip scans.** Journal of Bone and Mineral Research 1999, **14**:S369-S369.

225. Cuka S, Dvornik S, Drazenovi K, Mihi J: **Evaluation of the Dade Behring Dimension RxL clinical chemistry analyzer.** Clinical Laboratory 2001, **47**(1-2):35-40.

226. McAuley KA, Williams SM, Mann JI, Walker RJ, Lewis-Barned NJ, Temple LA, Duncan AW: **Diagnosing insulin resistance in the general population.** Diabetes Care 2001, **24**(3):460-464.

227. Fielding RA, Frontera WR, Hughes VA, Fisher EC, Evans W: **The reproducibility of the Bruce protocol exercise test for the determination of aerobic capacity in older women.** Med Sci Sports Exerc 1997, **29**(8):1109.

228. Heyward VH: Advanced Fitness Assessment and Exercise Prescription: 5th ed. Champaign, IL: Human Kinetics; 2006.
229. Wilborn CD, Kerksick CM, Campbell BI, Taylor LW, Marcello BM, Rasmussen CJ, Greenwood MC, Almada A, Kreider RB: **Effects of zinc magnesium aspartate (ZMA) supplementation on training adaptations and markers of anabolism and catabolism.** Journal of the International Society of Sports Nutrition 2004, **1**(2):12-20.
230. Baechle T ER: Essentials of Strength Training and Conditioning. Champaign, IL: Human Kinetics; 2005.
231. Brazier J, Harper R, Jones N, O'cathain A, Thomas K, Usherwood T, Westlake L: **Validating the SF-36 health survey questionnaire: new outcome measure for primary care.** BMJ: British Medical Journal 1992, **305**(6846):160.
232. Kosinski M, Keller SD, Hatoum HT, Kong SX, Ware Jr JE: **The SF-36 Health Survey as a generic outcome measure in clinical trials of patients with osteoarthritis and rheumatoid arthritis: tests of data quality, scaling assumptions and score reliability.** Med Care 1999, **37**(5):MS10-MS22.
233. Rosenberg M: Conceiving the Self. New York: Basic Books, 1979.
234. Hart EA, Leary MR, Rejeski WJ: **The measurement of social physique anxiety.** J Sport Exercise Psychol 1989, **11**(1):94-104.
235. Cash TF: User's Manual for the Multidimensional Body-Self Relations Questionnaire. Norfolk, VA: Old Dominion University, 2000.
236. Brinkworth GD, Noakes M, Keogh JB, Luscombe N, Wittert GA, Clifton PM: **Long-term effects of a high-protein, low-carbohydrate diet on weight control and cardiovascular risk markers in obese hyperinsulinemic subjects.** Int J Obes 2004, **28**(5):661-670.
237. Parker B, Noakes M, Luscombe N, Clifton P: **Effect of a high-protein, high-monounsaturated fat weight loss diet on glycemic control and lipid levels in type 2 diabetes.** Diabetes Care 2002, **25**(3):425-430.
238. Brinton EA, Eisenberg S, Breslow JL: **A low-fat diet decreases high density lipoprotein (HDL) cholesterol levels by decreasing HDL apolipoprotein transport rates.** J Clin Invest 1990, **85**:144-151.
239. Thompson PD, Jeffery RW, Wing RR, Wood PD: **Unexpected decrease in plasma high density lipoprotein cholesterol with weight loss.** Am J Clin Nutr 1979, **32**(10):2016-2021.

240. Ng TW, Watts GF, Barrett PHR, Rye K, Chan DC: **Effect of weight loss on LDL and HDL kinetics in the metabolic syndrome associations with changes in plasma retinol-binding protein-4 and adiponectin levels.** Diabetes Care 2007, **30**(11):2945-2950.

APPENDIX A

WAIST CIRCUMFERENCE RISK FACTOR TABLES

Table A.1: Body Composition Before and After Ten Weeks of Exercise and Dietary Intervention Based on Waist Circumference Status and Measured via DEXA Scan

Variable	Group	Baseline			10 Weeks			Group (SEM)			P-level
Scanned Mass (kg)	HP-LWC	71.6	±	8.3	68.6 [§]	±	8.1				T = 0.001
	HP-HWC	92.3	±	17.6	88.2 ^{c,§}	±	17.1				D = 0.11
	HC-LWC	72.3	±	9.4	70.0 [§]	±	9.1				WC = 0.001
	HC-HWC	86.1	±	14.1	82.8 ^{d,f,§}	±	13.5				D x WC = 0.018
	HP	89.2	±	18.1	85.3 [§]	±	17.5	80.2	±	1.1	T x D = 0.032
	HC	82.5	±	14.3	79.5 [§]	±	13.7	77.8	±	1.0	T x WC = 0.003
	LWC	72.0	±	9.0	69.4 ^{b,§}	±	8.7	70.6	±	1.3	T x D x WC = 0.82
	HWC	89.8	±	16.5	86.0 [§]	±	15.9	87.4 [‡]	±	0.7	
	Time	86.2	±	16.9	82.7 [*]	±	16.2				
Fat Mass (kg)	HP-LWC	30.0	±	4.9	27.3 [§]	±	5.3				T = 0.001
	HP-HWC	43.0	±	11.1	39.9 ^{c,§}	±	10.9				D = 0.21
	HC-LWC	31.1	±	5.6	29.4 [§]	±	5.7				WC = 0.001
	HC-HWC	39.2	±	9.1	36.6 ^{d,f,§}	±	8.8				D x WC = 0.006
	HP	41.1	±	11.4	38.0 [§]	±	11.2	35.0	±	0.7	T x D = 0.007
	HC	37.1	±	9.1	34.7 [§]	±	8.7	34.1	±	0.6	T x WC = 0.006
	LWC	30.6	±	5.3	28.5 ^{b,§}	±	5.6	29.4	±	0.8	T x D x WC = 0.43
	HWC	41.5	±	10.5	38.5 [§]	±	10.2	39.7 [‡]	±	0.4	
	Time	39.3	±	10.7	36.5 [*]	±	10.3				
Lean Mass (kg)	HP-LWC	39.9	±	4.8	39.6	±	4.3				T = 0.001
	HP-HWC	47.4	±	7.6	46.5 ^{c,§}	±	7.3				D = 0.035
	HC-LWC	39.5	±	5.0	38.9 [§]	±	4.7				WC = 0.001
	HC-HWC	45.1	±	6.3	44.5 ^{d,f,§}	±	6.0				D x WC = 0.19
	HP	46.3	±	7.7	45.5 [§]	±	7.4	43.4 [†]	±	0.5	T x D = 0.98
	HC	43.6	±	6.4	43.0 ^{a,§}	±	6.2	42.0	±	0.4	T x WC = 0.16
	LWC	39.7	±	4.9	39.2 ^{b,§}	±	4.5	39.5	±	0.6	T x D x WC = 0.14
	HWC	46.5	±	7.2	45.7 [§]	±	6.9	45.9 [‡]	±	0.3	
	Time	45.1	±	7.3	44.4 [*]	±	7.0				
Body Fat (%)	HP-LWC	42.1	±	4.0	40.2 [§]	±	4.4				T = 0.001
	HP-HWC	46.1	±	4.2	44.6 ^{c,§}	±	4.5				D = 0.43
	HC-LWC	43.1	±	3.9	42.1 ^{c,§}	±	4.0				WC = 0.001
	HC-HWC	45.3	±	4.1	43.8 ^{d,f,§}	±	4.3				D x WC = 0.007
	HP	45.5	±	4.4	43.9 [§]	±	4.7	43.3	±	0.3	T x D = 0.01
	HC	44.7	±	4.2	43.4 [§]	±	4.3	43.6	±	0.3	T x WC = 0.78
	LWC	42.7	±	4.0	41.3 ^{b,§}	±	4.3	41.9	±	0.4	T x D x WC = 0.015
	HWC	45.8	±	4.2	44.3 [§]	±	4.4	44.9 [‡]	±	0.2	
	Time	45.2	±	4.3	43.7 [*]	±	4.5				

HP = higher protein, HC = higher carbohydrate, LWC = low waist circumference, HWC = high waist circumference, T = time effect, D = diet effect, WC = waist circumference risk factor effect, D x WC = diet by waist circumference risk factor effect, T x D = time by diet effect, T x WC = time by waist circumference risk factor effect, T x D x WC = time by diet by waist circumference risk factor effect.

Values are means ± standard deviations (except group means are means ± standard error) from 55 participants in the HP-LWC group, 316 in the HP-HWC group, 77 in the HC-LWC group, 215 in the HC-HWC group, 371 in the HP total group, 292 in the HC total group, 131 in the LWC group, 531 in the HWC group, and 663 participants total.

* Significantly different than baseline, $p < 0.05$ (univariate). † Significant diet effect, $p < 0.05$ (univariate). ‡ Significant waist circumference effect, $p < 0.05$ (univariate). ^a Significantly different than HP group, $p < 0.05$ (post hoc LSD). ^b Significantly different than LWC group, $p < 0.05$ (post hoc LSD). ^c Significantly different than HP-LWC group, $p < 0.05$ (post hoc LSD). ^d Significantly different than HP-HWC group, $p < 0.05$ (post hoc LSD). ^e Significantly different than HC-LWC group, $p < 0.05$ (post hoc LSD). ^f Significantly different than baseline, $p < 0.05$ (post hoc LSD).

Table A.2: Body Composition Before and After Ten Weeks of Exercise and Dietary Intervention Based on Waist Circumference Status and Measured via Anthropometric Measurements

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Weight (kg)	HP-LWC	77.3	± 8.6	74.0 ^g	± 8.4				T = 0.001
	HP-HWC	99.0	± 18.5	94.5 ^{c,g}	± 17.9				D = 0.12
	HC-LWC	77.9	± 9.3	75.8 ^g	± 9.3				WC = 0.001
	HC-HWC	92.7	± 14.6	89.0 ^{d,f,g}	± 14.0				D x WC = 0.020
	HP	95.8	± 19.1	91.5 ^g	± 18.3	86.2	± 1.1	T x D = 0.004	
	HC	88.8	± 14.9	85.5 ^g	± 14.2	83.8	± 1.0	T x WC = 0.001	
	LWC	77.7	± 9.0	75.0 ^{b,g}	± 9.0	76.3	± 1.4	T x D x WC =0.60	
	HWC	96.4	± 17.3	92.3 ^g	± 16.7	93.8 [‡]	± 0.7		
Time	92.7	± 17.7	88.9 [*]	± 16.9					
Body Mass Index (kg/m ²)	HP-LWC	29.5	± 2.8	28.2 ^g	± 3.0				T = 0.001
	HP-HWC	37.1	± 6.4	35.4 ^{c,g}	± 6.2				D = 0.042
	HC-LWC	29.3	± 2.6	28.5 ^g	± 2.7				WC = 0.001
	HC-HWC	34.7	± 5.2	33.4 ^{d,f,g}	± 5.0				D x WC = 0.034
	HP	35.9	± 6.6	34.3 ^g	± 6.4	32.5 [†]	± 0.4	T x D =0.004	
	HC	33.3	± 5.2	32.1 ^g	± 5.0	31.5	± 0.4	T x WC = 0.001	
	LWC	29.4	± 2.7	28.4 ^{b,g}	± 2.9	28.9	± 0.5	T x D x WC = 0.61	
	HWC	36.1	± 6.1	34.6 ^g	± 5.8	35.1 [‡]	± 0.2		
Time	34.8	± 6.2	33.3	± 5.9					
Waist Circumference (cm)	HP-LWC	83.6	± 3.0	82.1	± 4.4				T = 0.001
	HP-HWC	104.4	± 12.1	100.0 ^{c,g}	± 12.4				D =0.045
	HC-LWC	83.1	± 3.7	82.5	± 5.3				WC = 0.001
	HC-HWC	100.3	± 10.1	96.2 ^{f,g}	± 10.5				D x WC = 0.053
	HP	101.3	± 13.5	97.3 ^g	± 13.2	92.5 [†]	± 0.7	T x D = 0.27	
	HC	95.8	± 11.7	92.6 ^{a,g}	± 11.2	90.5	± 0.7	T x WC = 0.001	
	LWC	83.3	± 3.4	82.3 ^{b,g}	± 4.9	82.8	± 0.9	T x D x WC = 0.62	
	HWC	102.8	± 11.5	98.4 ^g	± 11.8	100.2 [‡]	± 0.5		
Time	98.9	± 13.0	95.2 [*]	± 12.6					
Hip Circumference (cm)	HP-LWC	110.4	± 7.4	107.3 ^g	± 7.2				T = 0.001
	HP-HWC	124.9	± 13.6	121.7 ^{c,g}	± 13.4				D = 0.25
	HC-LWC	110.9	± 7.2	109.1 ^g	± 7.2				WC = 0.001
	HC-HWC	121.0	± 11.6	118.0 ^{d,f,g}	± 11.0				D x WC = 0.029
	HP	122.7	± 13.9	119.5 ^g	± 13.6	116.0	± 0.8	T x D = 0.12	
	HC	118.3	± 11.5	115.6 ^g	± 10.9	114.7	± 0.8	T x WC = 0.15	
	LWC	110.6	± 7.3	108.4 ^{b,g}	± 7.2	109.4	± 1.0	T x D x WC =0.23	
	HWC	123.3	± 13.0	120.2 ^g	± 12.6	121.4 [‡]	± 0.5		
Time	120.8	± 13.1	117.8 [*]	± 12.6					
HP = higher protein, HC = higher carbohydrate, LWC = low waist circumference, HWC = high waist circumference, T = time effect, D = diet effect, WC = waist circumference risk factor effect, D x WC = diet by waist circumference risk factor effect, T x D = time by diet effect, T x WC = time by waist circumference risk factor effect. Values are means ± standard deviations (except group means are means ± standard error) from 55 participants in the HP-LWC group, 316 in the HP-HWC group, 77 in the HC-LWC group, 215 in the HC-HWC group, 371 in the HP total group, 292 in the HC total group, 131 in the LWC group, 531 in the HWC group, and 663 participants total. * Significantly different than baseline, p < 0.05 (univariate). † Significant diet effect, p < 0.05 (univariate). ‡ Significant waist circumference effect, p < 0.05 (univariate). ^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than LWC group, p < 0.05 (post hoc LSD). ^c Significantly different than HP-LWC group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-HWC group, p < 0.05 (post hoc LSD). ^f Significantly different than HC-LWC group, p < 0.05 (post hoc LSD). ^g Significantly different than baseline, p <0.05 (post hoc LSD).									

Table A.3: Resting Energy Expenditure After Ten Weeks of Exercise and Dietary Intervention Based on Waist Circumference Status

Variable	Group	Baseline			10 Weeks			Group (SEM)			P-level
Resting Energy Expenditure (kcal/day)	HP-LWC	1450.6	±	182.5	1371.2 ^g	±	158.9				T = 0.001
	HP-HWC	1734.8	±	279.5	1673.6 ^{c,g}	±	287.6				D = 0.23
	HC-LWC	1459.1	±	207.9	1401.4 ^g	±	214.1				WC = 0.001
	HC-HWC	1646.0	±	243.1	1604.9 ^{d,f,g}	±	239.2				D x WC = 0.046
	HP	1697.4	±	285.2	1633.8 ^g	±	292.5	1557.6	±	18.2	T x D = 0.35
	HC	1600.7	±	248.0	1555.5 ^g	±	248.8	1527.9	±	16.4	T x WC = 0.44
	LWC	1455.6	±	197.0	1388.9 ^{b,g}	±	193.1	1420.6	±	22.2	T x D x WC = 0.97
	HWC	1699.0	±	268.7	1645.9 ^g	±	271.0	1664.8 [‡]	±	10.4	
Time	1655.2	±	273.7	1599.7 [*]	±	276.8					
HP = higher protein, HC = higher carbohydrate, LWC = low waist circumference, HWC = high waist circumference, T = time effect, D = diet effect, WC = waist circumference risk factor effect, D x WC = diet by waist circumference risk factor effect, T x D = time by diet effect, T x WC = time by waist circumference risk factor effect, T x D x WC = time by diet by waist circumference risk factor effect. Values are means ± standard deviations (except group means are means ± standard error) from 47 participants in the HP-LWC group, 310 in the HP-HWC group, 67 in the HC-LWC group, 209 in the HC-HWC group, 357 in the HP total group, 276 in the HC total group, 114 in the LWC group, 519 in the HWC group, and 633 participants total. * Significantly different than baseline, p < 0.05 (univariate). † Significant diet effect, p < 0.05 (univariate). ‡ Significant waist circumference effect, p < 0.05 (univariate). ^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than LWC group, p < 0.05 (post hoc LSD). ^c Significantly different than HP-LWC group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-HWC group, p < 0.05 (post hoc LSD). ^e Significantly different than HC-LWC group, p < 0.05 (post hoc LSD). ^f Significantly different than baseline, p < 0.05 (post hoc LSD).											

Table A.4: Resting Hemodynamic Measurements After Ten Weeks of Exercise and Dietary Intervention Based on Waist Circumference Status

Variable	Group	Baseline			10 Weeks			Group (SEM)			P-level
Resting Heart Rate (bpm)	HP-LWC	68.7	±	9.5	69.0	±	8.5				T = 0.001
	HP-HWC	72.6	±	10.4	68.9 ^g	±	9.9				D = 0.98
	HC-LWC	70.7	±	11.0	69.1	±	11.2				WC = 0.35
	HC-HWC	71.3	±	10.3	67.9 ^g	±	9.5				D x WC = 0.21
	HP	72.0	±	10.3	68.9 ^g	±	9.7	69.8	±	0.6	T x D = 0.45
	HC	71.2	±	10.5	68.2 ^g	±	9.9	69.8	±	0.6	T x WC = 0.005
	LWC	69.9	±	10.4	69.1	±	10.1	69.4	±	0.8	T x D x WC = 0.30
	HWC	72.1	±	10.4	68.5 ^g	±	9.7	70.2	±	0.4	
Time	71.6	±	10.4	68.6 [*]	±	9.8					
Resting Systolic Blood Pressure (mmHg)	HP-LWC	123.0	±	14.2	120.1	±	13.2				T = 0.001
	HP-HWC	126.0	±	14.8	123.5 ^g	±	13.8				D = 0.14
	HC-LWC	120.4	±	12.4	116.5 ^g	±	13.6				WC = 0.001
	HC-HWC	125.7	±	14.9	122.9 ^{f,g}	±	14.0				D x WC = 0.28
	HP	125.6	±	14.7	123.0 ^g	±	13.7	123.1	±	0.9	T x D = 0.67
	HC	124.3	±	14.4	121.2 ^g	±	14.1	121.4	±	0.8	T x WC = 0.62
	LWC	121.5	±	13.2	118.0 ^{b,g}	±	13.5	120.0	±	1.1	T x D x WC = 0.81
	HWC	125.9	±	14.8	123.2 ^g	±	13.8	124.5 [‡]	±	0.5	
Time	125.0	±	14.6	122.2 [*]	±	13.9					
Resting Diastolic Blood Pressure (mmHg)	HP-LWC	78.6	±	8.7	76.6	±	8.7				T = 0.001
	HP-HWC	81.7	±	9.3	80.1 ^{c,g}	±	8.7				D = 0.011
	HC-LWC	77.1	±	8.9	74.1 ^g	±	7.7				WC = 0.001
	HC-HWC	80.2	±	9.5	78.1 ^{d,f,g}	±	9.4				D x WC = 0.88
	HP	81.3	±	9.3	79.6 ^g	±	8.8	79.3 [†]	±	0.6	T x D = 0.46
	HC	79.3	±	9.4	77.0 ^{a,g}	±	9.2	77.3	±	0.5	T x WC = 0.55
	LWC	77.7	±	8.8	75.2 ^{b,g}	±	8.2	76.6	±	0.7	T x D x WC = 0.80
	HWC	81.1	±	9.4	79.3 ^g	±	9.1	80.0 [‡]	±	0.3	
Time	80.4	±	9.4	78.4 [*]	±	9.1					
HP = higher protein, HC = higher carbohydrate, LWC = low waist circumference, HWC = high waist circumference, T = time effect, D = diet effect, WC = waist circumference risk factor effect, D x WC = diet by waist circumference risk factor effect, T x D = time by diet effect, T x WC = time by waist circumference risk factor effect, T x D x WC = time by diet by waist circumference risk factor effect. Values are means ± standard deviations (except group means are means ± standard error) from 55 participants in the HP-LWC group, 316 in the HP-HWC group, 77 in the HC-LWC group, 215 in the HC-HWC group, 371 in the HP total group, 292 in the HC total group, 131 in the LWC group, 531 in the HWC group, and 663 participants total. [*] Significantly different than baseline, p < 0.05 (univariate). [†] Significant diet effect, p < 0.05 (univariate). [‡] Significant waist circumference effect, p < 0.05 (univariate). ^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than LWC group, p < 0.05 (post hoc LSD). ^c Significantly different than HP-LWC group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-HWC group, p < 0.05 (post hoc LSD). ^e Significantly different than HC-LWC group, p < 0.05 (post hoc LSD). ^f Significantly different than baseline, p < 0.05 (post hoc LSD).											

Table A.5: Fasting Blood Lipid Values After Ten Weeks of Exercise and Dietary Intervention Based on Waist Circumference Status

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Total Cholesterol (mmol/L)	HP-LWC	5.5	± 1.2	5.2 ^g	± 1.1				T = 0.001
	HP-HWC	5.2	± 0.9	5.0 ^g	± 0.9				D = 0.27
	HC-LWC	5.1	± 1.0	4.9 ^g	± 0.9				WC = 0.62
	HC-HWC	5.2	± 1.1	5.1 ^g	± 1.0				D x WC = 0.021
	HP	5.2	± 1.0	5.0 ^g	± 1.0	5.2	± 0.07		T x D = 0.35
	HC	5.2	± 1.0	5.0 ^g	± 1.0	5.1	± 0.06		T x WC = 0.25
	LWC	5.3	± 1.1	5.0 ^g	± 1.0	5.2	± 0.08		T x D x WC = 0.77
	HWC	5.2	± 1.0	5.0 ^g	± 1.0	5.1	± 0.04		
LDL (mmol/L)	HP-LWC	3.3	± 0.9	3.0 ^g	± 0.8				T = 0.001
	HP-HWC	3.1	± 0.7	2.9 ^g	± 0.7				D = 0.33
	HC-LWC	3.0	± 0.7	2.8 ^g	± 0.7				WC = 0.57
	HC-HWC	3.2	± 0.9	3.1 ^{f,g}	± 0.8				D x WC = 0.009
	HP	3.1	± 0.8	3.0 ^g	± 0.8	3.1	± 0.05		T x D = 0.45
	HC	3.1	± 0.8	3.0 ^g	± 0.8	3.0	± 0.05		T x WC = 0.14
	LWC	3.1	± 0.8	2.9 ^g	± 0.8	3.0	± 0.06		T x D x WC = 0.69
	HWC	3.1	± 0.8	3.0 ^g	± 0.8	3.1	± 0.03		
HDL (mmol/L)	HP-LWC	1.5	± 0.3	1.4 ^g	± 0.3				T = 0.001
	HP-HWC	1.4	± 0.3	1.3 ^{c,g}	± 0.3				D = 0.76
	HC-LWC	1.5	± 0.4	1.4 ^g	± 0.3				WC = 0.001
	HC-HWC	1.4	± 0.3	1.3 ^{f,g}	± 0.3				D x WC = 0.88
	HP	1.4	± 0.3	1.3 ^g	± 0.3	1.4	± 0.02		T x D = 0.88
	HC	1.4	± 0.3	1.3 ^g	± 0.3	1.4	± 0.02		T x WC = 0.98
	LWC	1.5	± 0.4	1.4 ^{b,g}	± 0.3	1.5	± 0.03		T x D x WC = 0.87
	HWC	1.4	± 0.3	1.3 ^g	± 0.3	1.3 [‡]	± 0.01		
Triglycerides (mmol/L)	HP-LWC	1.4	± 0.7	1.3	± 0.9				T = 0.008
	HP-HWC	1.6	± 0.9	1.5 ^g	± 0.8				D = 0.62
	HC-LWC	1.3	± 0.7	1.3	± 0.8				WC = 0.005
	HC-HWC	1.6	± 0.8	1.5 ^f	± 0.7				D x WC = 0.63
	HP	1.6	± 0.8	1.5 ^g	± 0.8	1.5	± 0.05		T x D = 0.11
	HC	1.5	± 0.7	1.5	± 0.7	1.4	± 0.05		T x WC = 0.36
	LWC	1.4	± 0.7	1.3 ^b	± 0.8	1.4	± 0.06		T x D x WC = 0.92
	HWC	1.6	± 0.8	1.5 ^g	± 0.8	1.6 [‡]	± 0.03		
TC/HDL ratio	HP-LWC	3.8	± 0.9	3.7	± 1.0				T = 0.38
	HP-HWC	3.9	± 0.9	4.0	± 0.9				D = 0.66
	HC-LWC	3.6	± 0.9	3.6	± 0.9				WC = 0.001
	HC-HWC	4.0	± 1.0	4.1 ^{f,g}	± 1.1				D x WC = 0.15
	HP	3.9	± 0.9	3.9	± 0.9	3.9	± 0.07		T x D = 0.50
	HC	3.9	± 1.0	4.0	± 1.1	3.8	± 0.06		T x WC = 0.14
	LWC	3.7	± 0.9	3.7 ^b	± 0.9	3.7	± 0.08		T x D x WC = 0.89
	HWC	4.0	± 1.0	4.0 ^g	± 1.0	4.0 [‡]	± 0.04		
HP = higher protein, HC = higher carbohydrate, LWC = low waist circumference, HWC = high waist circumference, T = time effect, D = diet effect, WC = waist circumference risk factor effect, D x WC = diet by waist circumference risk factor effect, T x D = time by diet effect, T x WC = time by waist circumference risk factor effect, T x D x WC = time by diet by waist circumference risk factor effect. Values are means ± standard deviations (except group means are means ± standard error) from 55 participants in the HP-LWC group, 316 in the HP-HWC group, 77 in the HC-LWC group, 215 in the HC-HWC group, 371 in the HP total group, 292 in the HC total group, 131 in the LWC group, 531 in the HWC group, and 663 participants total. * Significantly different than baseline, p < 0.05 (univariate). † Significant diet effect, p < 0.05 (univariate). ‡ Significant waist circumference effect, p < 0.05 (univariate). ^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than LWC group, p < 0.05 (post hoc LSD). ^c Significantly different than HP-LWC group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-HWC group, p < 0.05 (post hoc LSD). ^e Significantly different than HC-LWC group, p < 0.05 (post hoc LSD). ^f Significantly different than baseline, p < 0.05 (post hoc LSD).									

Table A.6: Fasting Glucose and Insulin Values After Ten Weeks of Exercise and Dietary Intervention Based on Waist Circumference Status

Variable	Group	Baseline			10 Weeks			Group (SEM)			P-level
Glucose (mmol/L)	HP-LWC	5.6	±	1.0	5.4	±	1.1				T = 0.09
	HP-HWC	5.7	±	1.3	5.6 ^e	±	1.2				D = 0.36
	HC-LWC	5.3	±	0.5	5.4	±	0.6				WC = 0.022
	HC-HWC	5.7	±	1.5	5.6	±	1.1				D x WC = 0.46
	HP	5.7	±	1.2	5.6 ^e	±	1.2	5.6	±	0.08	T x D = 0.18
	HC	5.6	±	1.3	5.5	±	1.0	5.5	±	0.07	T x WC = 0.51
	LWC	5.4	±	0.7	5.4 ^b	±	0.8	5.4	±	0.10	T x D x WC = 0.20
	HWC	5.7	±	1.4	5.6 ^e	±	1.2	5.7 [‡]	±	0.05	
	Time	5.7	±	1.3	5.6	±	1.1				
Insulin (uIU/mL)	HP-LWC	5.3	±	6.3	7.2	±	10.0				T = 0.20
	HP-HWC	3.7	±	9.2	4.0	±	10.1				D = 0.035
	HC-LWC	5.5	±	4.8	8.5 ^e	±	12.5				WC = 0.94
	HC-HWC	10.0	±	15.8	8.4 ^d	±	11.7				D x WC = 0.12
	HP	4.0	±	8.8	4.5	±	10.1	5.0 [†]	±	1.06	T x D = 0.80
	HC	8.3	±	12.8	8.4	±	12.0	8.1	±	0.98	T x WC = 0.023
	LWC	5.4	±	5.4	8.0 ^e	±	11.6	6.6	±	1.24	T x D x WC = 0.28
	HWC	5.8	±	12.2	5.5	±	10.8	6.5	±	0.75	
	Time	5.7	±	10.8	6.1	±	11.1				
Calculated HOMA	HP-LWC	1.5	±	1.9	2.0	±	3.7				T = 0.36
	HP-HWC	1.0	±	2.4	1.0	±	2.7				D = 0.055
	HC-LWC	1.3	±	1.2	2.3	±	4.1				WC = 0.95
	HC-HWC	2.9	±	5.7	2.2 ^d	±	3.1				D x WC = 0.08
	HP	1.0	±	2.3	1.2	±	2.9	1.4	±	0.31	T x D = 0.71
	HC	2.3	±	4.6	2.2	±	3.5	2.2	±	0.28	T x WC = 0.031
	LWC	1.4	±	1.5	2.2	±	3.9	1.8	±	0.36	T x D x WC = 0.23
	HWC	1.6	±	3.9	1.4	±	2.9	1.8	±	0.22	
	Time	1.6	±	3.5	1.6	±	3.2				

HP = higher protein, HC = higher carbohydrate, LWC = low waist circumference, HWC = high waist circumference, T = time effect, D = diet effect, WC = waist circumference risk factor effect, D x WC = diet by waist circumference risk factor effect, T x D = time by diet effect, T x WC = time by waist circumference risk factor effect, T x D x WC = time by diet by waist circumference risk factor effect.

Glucose values are means ± standard deviations (except group means are means ± standard error) from 55 participants in the HP-LWC group, 316 in the HP-HWC group, 77 in the HC-LWC group, 215 in the HC-HWC group, 371 in the HP total group, 292 in the HC total group, 131 in the LWC group, 531 in the HWC group, and 663 participants total. Insulin and HOMA values are means ± standard deviations (except group means are means ± standard error) from 25 participants in the HP-LWC group, 124 in the HP-HWC group, 40 in the HC-LWC group, 63 in the HC-HWC group, 149 in the HP total group, 103 in the HC total group, 65 in the LWC group, 187 in the HWC group, and 252 participants total.

* Significantly different than baseline, $p < 0.05$ (univariate). † Significant diet effect, $p < 0.05$ (univariate). ‡ Significant waist circumference effect, $p < 0.05$ (univariate). ^a Significantly different than HP group, $p < 0.05$ (post hoc LSD). ^b Significantly different than LWC group, $p < 0.05$ (post hoc LSD). ^c Significantly different than HP-LWC group, $p < 0.05$ (post hoc LSD). ^d Significantly different than HP-HWC group, $p < 0.05$ (post hoc LSD). ^e Significantly different than HC-LWC group, $p < 0.05$ (post hoc LSD). ^f Significantly different than baseline, $p < 0.05$ (post hoc LSD).

Table A.7: Fitness Parameters After Ten Weeks of Exercise and Dietary Intervention Based on Waist Circumference Status

Variable	Group	Baseline			10 Weeks			Group (SEM)			P-level
Bench Press Max Strength (1 RM / kg body weight)	HP-LWC	30.0	±	8.3	32.2 ^a	±	8.5				T = 0.001
	HP-HWC	31.1	±	7.5	33.0 ^b	±	7.3				D = 0.17
	HC-LWC	28.3	±	6.7	30.4 ^b	±	7.0				WC = 0.06
	HC-HWC	30.3	±	8.1	32.7 ^{f,g}	±	8.3				D x WC = 0.46
	HP	30.9	±	7.6	32.9 ^b	±	7.4	31.6	±	0.6	T x D = 0.74
	HC	29.9	±	7.8	32.2 ^b	±	8.1	30.5	±	0.6	T x WC = 0.91
	LWC	29.1	±	7.4	31.2 ^b	±	7.7	30.3	±	0.7	T x D x WC = 0.61
	HWC	30.8	±	7.7	32.9 ^b	±	7.7	31.8 [‡]	±	0.3	
	Time	30.5	±	7.7	32.6 [*]	±	7.7				
Bench Press Lift Volume (kg)	HP-LWC	163.3	±	65.6	175.1	±	69.0				T = 0.51
	HP-HWC	182.6	±	81.3	182.9	±	82.2				D = 0.91
	HC-LWC	163.9	±	65.9	172.0	±	93.8				WC = 0.040
	HC-HWC	189.4	±	88.3	182.4	±	75.7				D x WC = 0.78
	HP	180.3	±	79.7	182.0	±	80.7	176.0	±	5.7	T x D = 0.58
	HC	183.8	±	84.5	180.1	±	79.9	176.9	±	5.1	T x WC = 0.18
	LWC	163.7	±	65.5	173.3	±	83.8	168.6	±	7.0	T x D x WC = 0.86
	HWC	185.3	±	84.2	182.7	±	79.6	184.3 [‡]	±	3.1	
	Time	181.8	±	81.8	181.2	±	80.3				
Leg Press Max Strength (1 RM / kg body weight)	HP-LWC	139.0	±	32.0	148.6 ^b	±	35.8				T = 0.001
	HP-HWC	167.6	±	48.9	184.4 ^{c,g}	±	52.7				D = 0.78
	HC-LWC	138.4	±	41.5	153.2 ^b	±	44.9				WC = 0.001
	HC-HWC	164.0	±	51.9	178.0 ^{f,g}	±	56.7				D x WC = 0.52
	HP	164.1	±	48.1	180.1 ^b	±	52.3	159.9	±	4.0	T x D = 0.72
	HC	158.4	±	50.8	172.5 ^b	±	55.2	158.4	±	3.7	T x WC = 0.32
	LWC	138.6	±	37.6	151.3 ^{b,g}	±	41.2	144.8	±	5.0	T x D x WC = 0.22
	HWC	166.1	±	50.1	181.8 ^b	±	54.4	173.5 [‡]	±	2.2	
	Time	161.7	±	49.3	176.8 [*]	±	53.6				
Leg Press Lift Volume (kg)	HP-LWC	1568.3	±	798.9	1514.1	±	695.5				T = 0.96
	HP-HWC	1856.5	±	985.4	1990.5 ^{c,g}	±	1108.8				D = 0.10
	HC-LWC	1461.2	±	836.5	1401.3	±	742.6				WC = 0.001
	HC-HWC	1725.0	±	865.2	1716.3 ^{d,f}	±	931.5				D x WC = 0.63
	HP	1821.8	±	968.3	1933.1	±	1077.9	1732.4	±	70.8	T x D = 0.46
	HC	1667.3	±	864.3	1647.4	±	901.7	1575.9	±	64.2	T x WC = 0.23
	LWC	1506.5	±	818.3	1449.0 ^b	±	721.5	1486.2	±	87.3	T x D x WC = 0.50
	HWC	1803.8	±	940.4	1880.6	±	1049.0	1822.1 [‡]	±	38.8	
	Time	1755.4	±	927.5	1810.3	±	1015.1				
Peak VO₂ (mL/kg/min)	HP-LWC	23.2	±	4.7	24.1 ^b	±	5.0				T = 0.001
	HP-HWC	19.5	±	4.3	21.6 ^{c,g}	±	4.3				D = 0.74
	HC-LWC	21.8	±	4.5	24.5 ^b	±	4.8				WC = 0.001
	HC-HWC	20.0	±	4.0	22.6 ^{d,f,g}	±	4.6				D x WC = 0.12
	HP	20.0	±	4.5	21.9 ^b	±	4.5	22.1	±	0.3	T x D = 0.001
	HC	20.5	±	4.2	23.1 ^b	±	4.7	22.2	±	0.3	T x WC = 0.10
	LWC	22.4	±	4.6	24.3 ^{b,g}	±	4.9	23.4	±	0.4	T x D x WC = 0.044
	HWC	19.7	±	4.2	22.0 ^b	±	4.5	20.9 [‡]	±	0.2	
	Time	20.2	±	4.4	22.4 [*]	±	4.6				

HP = higher protein, HC = higher carbohydrate, LWC = low waist circumference, HWC = high waist circumference, T = time effect, D = diet effect, WC = waist circumference risk factor effect, D x WC = diet by waist circumference risk factor effect, T x D = time by diet effect, T x WC = time by waist circumference risk factor effect, T x D x WC = time by diet by waist circumference risk factor effect.

Values are means ± standard deviations (except group means are means ± standard error) from 55 participants in the HP-LWC group, 316 in the HP-HWC group, 77 in the HC-LWC group, 215 in the HC-HWC group, 371 in the HP total group, 292 in the HC total group, 131 in the LWC group, 531 in the HWC group, and 663 participants total.

* Significantly different than baseline, $p < 0.05$ (univariate). † Significant diet effect, $p < 0.05$ (univariate). ‡ Significant waist circumference effect, $p < 0.05$ (univariate). ^a Significantly different than HP group, $p < 0.05$ (post hoc LSD). ^b Significantly different than LWC group, $p < 0.05$ (post hoc LSD). ^c Significantly different than HP-LWC group, $p < 0.05$ (post hoc LSD). ^d Significantly different than HP-HWC group, $p < 0.05$ (post hoc LSD). ^e Significantly different than HC-LWC group, $p < 0.05$ (post hoc LSD). ^f Significantly different than baseline, $p < 0.05$ (post hoc LSD).

Table A.8: Changes in SF36 - Quality of Life Inventory Values After Ten Weeks of Exercise and Dietary Intervention Based on Waist Circumference Status

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Physical Functioning	HP-LWC	81.6	± 32.6	88.8	± 26.4				T = 0.001
	HP-HWC	74.5	± 29.1	79.8 ^g	± 24.2				D = 0.70
	HC-LWC	77.6	± 15.9	84.8 ^g	± 23.7				WC = 0.10
	HC-HWC	75.8	± 23.3	81.9 ^g	± 18.5				D x WC = 0.35
	HP	75.7	± 29.7	81.3 ^g	± 24.7	81.2	± 2.4		T x D = 0.90
	HC	76.4	± 21.2	82.8 ^g	± 20.2	80.0	± 2.0		T x WC = 0.65
	LWC	79.0	± 23.3	86.2 ^g	± 24.6	83.2	± 2.7		T x D x WC = 0.91
	HWC	75.1	± 26.8	80.7 ^g	± 21.9	78.0	± 1.5		
	Time	76.0	± 26.0	82.0*	± 22.7				
Role Physical	HP-LWC	179.8	± 143.7	182.1	± 153.3				T = 0.63
	HP-HWC	134.8	± 135.6	139.4	± 146.5				D = 0.001
	HC-LWC	228.2	± 137.3	223.1	± 151.1				WC = 0.85
	HC-HWC	258.0	± 144.3	265.7 ^d	± 144.1				D x WC = 0.045
	HP	142.2	± 137.5	146.4	± 148.0	159.0†	± 15.2		T x D = 0.82
	HC	248.6	± 142.3	252.3 ^a	± 147.1	243.7	± 12.8		T x WC = 0.44
	LWC	210.4	± 140.6	208.0	± 152.1	203.3	± 17.5		T x D x WC = 0.60
	HWC	187.2	± 151.9	193.1	± 158.1	199.5	± 9.5		
	Time	192.6	± 149.4	196.6	± 156.5				
Bodily Pain	HP-LWC	60.8	± 20.6	67.0	± 21.8				T = 0.006
	HP-HWC	58.7	± 18.7	62.4 ^g	± 20.5				D = 0.76
	HC-LWC	62.5	± 21.0	65.8	± 20.8				WC = 0.31
	HC-HWC	61.8	± 21.8	62.2	± 22.0				D x WC = 0.82
	HP	59.1	± 19.0	63.1 ^g	± 20.7	62.2	± 2.1		T x D = 0.22
	HC	62.0	± 21.4	63.3	± 21.6	63.0	± 1.7		T x WC = 0.28
	LWC	61.9	± 20.7	66.2 ^g	± 21.0	64.0	± 2.4		T x D x WC = 0.93
	HWC	60.0	± 20.1	62.3	± 21.1	61.3	± 1.3		
	Time	60.5	± 20.2	63.2*	± 21.1				
General Health	HP-LWC	63.2	± 25.9	67.8 ^g	± 26.4				T = 0.001
	HP-HWC	47.8	± 28.5	52.2 ^{c,g}	± 28.4				D = 0.01
	HC-LWC	65.8	± 21.8	69.2	± 21.8				WC = 0.020
	HC-HWC	64.8	± 22.7	68.0 ^{d,g}	± 22.4				D x WC = 0.044
	HP	50.4	± 28.6	54.8 ^g	± 28.6	57.8†	± 2.7		T x D = 0.48
	HC	65.1	± 22.3	68.4 ^{a,g}	± 22.1	66.9	± 2.3		T x WC = 0.92
	LWC	64.8	± 23.2	68.7 ^{b,g}	± 23.4	66.5	± 3.1		T x D x WC = 0.96
	HWC	55.0	± 27.5	58.9 ^g	± 27.2	58.2‡	± 1.7		
	Time	57.3	± 26.8	61.2*	± 26.6				
Vital	HP-LWC	45.7	± 22.0	51.1	± 22.8				T = 0.001
	HP-HWC	40.4	± 49.5	48.7 ^g	± 51.0				D = 0.19
	HC-LWC	50.7	± 18.0	56.8 ^g	± 18.2				WC = 0.63
	HC-HWC	50.2	± 18.8	55.0 ^g	± 18.8				D x WC = 0.79
	HP	41.3	± 46.1	49.1 ^g	± 47.5	46.5	± 3.9		T x D = 0.48
	HC	50.4	± 18.5	55.5 ^g	± 18.6	53.2	± 3.3		T x WC = 0.72
	LWC	48.8	± 19.6	54.7 ^g	± 20.0	51.0	± 4.5		T x D x WC = 0.31
	HWC	44.6	± 39.7	51.4 ^g	± 40.6	48.6	± 2.4		
	Time	45.6	± 36.0	52.2*	± 36.8				

Table A.8: Continued

Variable	Group	Baseline		10 Weeks		Group (SEM)		P-level
Social Functioning	HP-LWC	58.2	± 28.3	60.3	± 25.8			T = 0.10
	HP-HWC	43.9	± 24.7	48.8 ^{c,g}	± 26.6			D = 0.08
	HC-LWC	56.4	± 20.5	56.2	± 21.5			WC = 0.24
	HC-HWC	60.6	± 26.7	61.9 ^d	± 25.7			D x WC = 0.009
	HP	46.2	± 25.7	50.7	± 26.7	52.8	± 2.6	T x D = 0.23
	HC	59.3	± 24.9	60.1	± 24.5	58.8	± 2.2	T x WC = 0.37
	LWC	57.1	± 23.5	57.7	± 23.1	57.8	± 3.0	T x D x WC = 0.81
	HWC	51.0	± 26.8	54.4 ^e	± 26.9	53.8	± 1.6	
	Time	52.4	± 26.1	55.2	± 26.1			
Role Emotional	HP-LWC	216.7	± 131.8	230.6	± 138.8			T = 0.47
	HP-HWC	218.6	± 128.7	240.2	± 141.4			D = 0.003
	HC-LWC	248.3	± 130.6	260.5	± 148.2			WC = 0.09
	HC-HWC	321.6	± 208.8	302.6 ^d	± 125.4			D x WC = 0.17
	HP	218.3	± 128.7	238.6	± 140.6	226.5 [†]	± 14.5	T x D = 0.28
	HC	298.6	± 190.3	289.4 ^a	± 133.9	283.2	± 12.2	T x WC = 0.55
	LWC	236.7	± 131.0	249.5	± 144.5	239.0	± 16.6	T x D x WC = 0.33
	HWC	262.4	± 174.7	266.7	± 138.1	270.7	± 9.0	
	Time	256.3	± 165.6	262.7	± 139.6			
Mental Health	HP-LWC	61.0	± 16.6	68.1 ^g	± 13.3			T = 0.001
	HP-HWC	55.7	± 15.2	64.6 ^e	± 15.7			D = 0.20
	HC-LWC	61.7	± 17.1	70.9 ^e	± 13.9			WC = 0.025
	HC-HWC	59.6	± 13.0	66.1 ^g	± 11.0			D x WC = 0.78
	HP	56.5	± 15.5	65.1 ^g	± 15.3	62.3	± 1.4	T x D = 0.94
	HC	60.3	± 14.4	67.6 ^g	± 12.1	64.6	± 1.1	T x WC = 0.83
	LWC	61.5	± 16.8	69.9 ^{b,g}	± 13.6	65.4	± 1.6	T x D x WC = 0.30
	HWC	57.3	± 14.4	65.2 ^e	± 13.9	61.5 [‡]	± 0.8	
	Time	58.3	± 15.1	66.3 [*]	± 13.9			

HP = higher protein, HC = higher carbohydrate, LWC = low waist circumference, HWC = high waist circumference, T = time effect, D = diet effect, WC = waist circumference risk factor effect, D x WC = diet by waist circumference risk factor effect, T x D = time by diet effect, T x WC = time by waist circumference risk factor effect, T x D x WC = time by diet by waist circumference risk factor effect.

Values are means ± standard deviations (except group means are means ± standard error) from 25 participants in the HP-LWC group, 127 in the HP-HWC group, 43 in the HC-LWC group, 94 in the HC-HWC group, 152 in the HP total group, 137 in the HC total group, 68 in the LWC group, 221 in the HWC group, and 289 participants total.

* Significantly different than baseline, $p < 0.05$ (univariate). † Significant diet effect, $p < 0.05$ (univariate). ‡ Significant waist circumference effect, $p < 0.05$ (univariate). ^a Significantly different than HP group, $p < 0.05$ (post hoc LSD). ^b Significantly different than LWC group, $p < 0.05$ (post hoc LSD). ^c Significantly different than HP-LWC group, $p < 0.05$ (post hoc LSD). ^d Significantly different than HP-HWC group, $p < 0.05$ (post hoc LSD). ^e Significantly different than HC-LWC group, $p < 0.05$ (post hoc LSD). ^f Significantly different than baseline, $p < 0.05$ (post hoc LSD).

Table A.9: Changes in Body Image Evaluation After Ten Weeks of Exercise and Dietary Intervention Based on Waist Circumference Status

Variable	Group	Baseline	10 Weeks	Group (SEM)	P-level
Appearance Evaluation	HP-LWC	2.6 ± 0.6	3.2 ^g ± 0.7		T = 0.001
	HP-HWC	2.4 ± 0.6	2.7 ^{c,g} ± 0.6		D = 0.27
	HC-LWC	2.5 ± 0.7	2.9 ^{c,g} ± 0.7		WC = 0.001
	HC-HWC	2.4 ± 0.6	2.8 ^g ± 0.7		D x WC = 0.037
	HP	2.4 ± 0.6	2.7 ^g ± 0.7	2.7 ± 0.05	T x D = 0.42
	HC	2.4 ± 0.6	2.8 ^g ± 0.7	2.6 ± 0.05	T x WC = 0.004
	LWC	2.5 ± 0.6	3.0 ^{b,g} ± 0.7	2.8 ± 0.06	T x D x WC = 0.13
	HWC	2.4 ± 0.6	2.7 ^g ± 0.7	2.6 [‡] ± 0.03	
	Time	2.4 ± 0.6	2.8 [*] ± 0.7		
Appearance Orientation	HP-LWC	4.0 ± 0.7	3.9 ± 0.7		T = 0.16
	HP-HWC	4.2 ± 0.9	4.2 ± 0.9		D = 0.24
	HC-LWC	3.9 ± 0.8	3.9 ± 0.8		WC = 0.20
	HC-HWC	4.0 ± 0.8	3.9 ^d ± 0.9		D x WC = 0.25
	HP	4.1 ± 0.9	4.1 ± 0.9	4.0 ± 0.07	T x D = 0.90
	HC	3.9 ± 0.8	3.9 ± 0.9	3.9 ± 0.07	T x WC = 0.56
	LWC	3.9 ± 0.8	3.9 ± 0.8	3.9 ± 0.09	T x D x WC = 0.43
	HWC	4.1 ± 0.8	4.1 ± 0.9	4.0 ± 0.05	
	Time	4.1 ± 0.8	4.0 ± 0.9		
Body Area Satisfaction	HP-LWC	2.4 ± 0.7	2.7 ^g ± 0.8		T = 0.001
	HP-HWC	2.0 ± 0.8	2.2 ^{c,g} ± 0.8		D = 0.23
	HC-LWC	2.3 ± 0.8	2.7 ^g ± 0.8		WC = 0.001
	HC-HWC	2.2 ± 0.7	2.5 ^{d,g} ± 0.8		D x WC = 0.09
	HP	2.0 ± 0.8	2.3 ^g ± 0.8	2.3 ± 0.07	T x D = 0.68
	HC	2.2 ± 0.7	2.5 ^g ± 0.8	2.4 ± 0.06	T x WC = 0.29
	LWC	2.3 ± 0.7	2.7 ^{b,g} ± 0.8	2.5 ± 0.08	T x D x WC = 0.38
	HWC	2.1 ± 0.7	2.3 ^g ± 0.8	2.2 [‡] ± 0.04	
	Time	2.1 ± 0.7	2.4 [*] ± 0.8		
Overweight Preoccupation	HP-LWC	2.7 ± 0.8	3.2 ^g ± 0.7		T = 0.001
	HP-HWC	2.9 ± 0.7	3.4 ^g ± 0.7		D = 0.95
	HC-LWC	2.9 ± 0.7	3.3 ^g ± 0.7		WC = 0.15
	HC-HWC	2.9 ± 0.7	3.3 ^g ± 0.7		D x WC = 0.25
	HP	2.9 ± 0.7	3.4 ^g ± 0.7	3.1 ± 0.06	T x D = 0.31
	HC	2.9 ± 0.7	3.3 ^g ± 0.7	3.1 ± 0.05	T x WC = 0.70
	LWC	2.8 ± 0.8	3.2 ^g ± 0.7	3.0 ± 0.07	T x D x WC = 0.80
	HWC	2.9 ± 0.7	3.4 ^g ± 0.7	3.1 ± 0.03	
	Time	2.9 ± 0.7	3.3 [*] ± 0.7		
Self-Classified Weight	HP-LWC	3.9 ± 0.7	3.8 ± 0.4		T = 0.045
	HP-HWC	4.3 ± 0.7	4.2 ^c ± 0.7		D = 0.97
	HC-LWC	4.0 ± 0.6	3.9 ± 0.5		WC = 0.001
	HC-HWC	4.2 ± 0.7	4.1 ^g ± 0.8		D x WC = 0.18
	HP	4.2 ± 0.7	4.2 ± 0.7	4.1 ± 0.05	T x D = 0.87
	HC	4.2 ± 0.7	4.0 ± 0.7	4.1 ± 0.04	T x WC = 0.86
	LWC	4.0 ± 0.6	3.9 ^b ± 0.5	3.9 ± 0.06	T x D x WC = 0.55
	HWC	4.3 ± 0.7	4.2 ^g ± 0.7	4.2 [‡] ± 0.03	
	Time	4.2 ± 0.7	4.1 [*] ± 0.7		

Table A.9: Continued

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Rosenberg Self Esteem	HP-LWC	26.1	± 3.8	26.0	± 3.7				T = 0.10
	HP-HWC	25.6	± 4.0	26.4 ^g	± 3.9				D = 0.77
	HC-LWC	25.8	± 4.0	26.4	± 4.2				WC = 0.60
	HC-HWC	25.7	± 3.9	25.7	± 3.9				D x WC = 0.63
	HP	25.7	± 3.9	26.3	± 3.8	26.0	± 0.32	T x D = 0.97	
	HC	25.7	± 3.9	25.9	± 4.0	25.9	± 0.28	T x WC = 0.72	
	LWC	25.9	± 3.9	26.3	± 4.0	26.1	± 0.37	T x D x WC = 0.07	
	HWC	25.7	± 3.9	26.1 ^h	± 3.9	25.8	± 0.20		
Time	25.7	± 3.9	26.2	± 3.9					
Social Physique Anxiety	HP-LWC	29.5	± 5.1	29.7	± 4.7				T = 0.79
	HP-HWC	31.6	± 6.4	31.6	± 6.4				D =0.40
	HC-LWC	31.0	± 5.5	30.8	± 4.9				WC = 0.047
	HC-HWC	31.2	± 6.2	31.5	± 6.6				D x WC = 0.19
	HP	31.3	± 6.3	31.4	± 6.2	30.6	± 0.45	T x D = 0.87	
	HC	31.1	± 6.0	31.3	± 6.1	31.1	± 0.40	T x WC = 0.83	
	LWC	30.4	± 5.4	30.4	± 4.8	30.2	± 0.53	T x D x WC = 0.65	
	HWC	31.4	± 6.3	31.6	± 6.5	31.4 [‡]	± 0.28		
Time	31.2	± 6.1	31.3	± 6.2					
HP = higher protein, HC = higher carbohydrate, LWC = low waist circumference, HWC = high waist circumference, T = time effect, D = diet effect, WC = waist circumference risk factor effect, D x WC = diet by waist circumference risk factor effect, T x D = time by diet effect, T x WC = time by waist circumference risk factor effect. Values are means ± standard deviations (except group means are means ± standard error) from 37 participants in the HP-LWC group, 219 in the HP-HWC group, 60 in the HC-LWC group, 135 in the HC-HWC group, 256 in the HP total group, 195 in the HC total group, 97 in the LWC group, 354 in the HWC group, and 451 participants total. * Significantly different than baseline, p < 0.05 (univariate). † Significant diet effect, p < 0.05 (univariate). ‡ Significant waist circumference effect, p < 0.05 (univariate). ^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than LWC group, p < 0.05 (post hoc LSD). ^c Significantly different than HP-LWC group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-HWC group, p < 0.05 (post hoc LSD). ^e Significantly different than HC-LWC group, p < 0.05 (post hoc LSD). ^f Significantly different than baseline, p <0.05 (post hoc LSD).									

APPENDIX B

TRIGLYCERIDE RISK FACTOR TABLES

Table B.1: Body Composition Before and After Ten Weeks of Exercise and Dietary Intervention Based on Triglyceride Status and Measured via DEXA Scan

Variable	Group	Baseline			10 Weeks			Group (SEM)			P-level
Scanned Mass (kg)	HP-LTG	90.5	±	18.6	86.4 [§]	±	18.0				T = 0.001
	HP-HTG	86.8	±	16.7	83.2 [§]	±	16.3				D = 0.001
	HC-LTG	82.0	±	14.8	79.0 ^{c,§}	±	14.2				TG = 0.45
	HC-HTG	83.5	±	13.3	80.4 [§]	±	12.6				D x TG = 0.07
	HP	89.2	±	18.1	85.3 [§]	±	17.5	86.7 [†]	±	0.9	T x D = 0.007
	HC	82.5	±	14.3	79.5 ^{a,§}	±	13.7	81.2	±	1.0	T x TG = 0.52
	LTG	86.7	±	17.6	83.1 [§]	±	16.8	84.5	±	0.8	T x D x TG = 0.22
	HTG	85.3	±	15.4	82.0 [§]	±	14.8	83.5	±	1.1	
	Time	86.2	±	16.9	82.7 [*]	±	16.2				
Fat Mass (kg)	HP-LTG	41.7	±	11.7	38.6 [§]	±	11.4				T = 0.001
	HP-HTG	39.8	±	10.9	36.9 [§]	±	10.6				D = 0.001
	HC-LTG	37.0	±	9.6	34.6 ^{c,§}	±	9.1				TG = 0.35
	HC-HTG	37.2	±	8.0	34.8 [§]	±	7.7				D x TG = 0.35
	HP	41.1	±	11.4	38.0 [§]	±	11.2	39.3 [†]	±	0.6	T x D = 0.004
	HC	37.1	±	9.1	34.7 ^{a,§}	±	8.7	35.9	±	0.6	T x TG = 0.58
	LTG	39.7	±	11.0	36.8 [§]	±	10.7	38.0	±	0.5	T x D x TG = 0.61
	HTG	38.7	±	9.8	36.0 [§]	±	9.5	37.2	±	0.7	
	Time	39.3	±	10.7	36.5 [*]	±	10.3				
Lean Mass (kg)	HP-LTG	46.9	±	8.1	46.0 [§]	±	7.7				T = 0.001
	HP-HTG	45.1	±	6.9	44.5 [§]	±	6.7				D = 0.001
	HC-LTG	43.2	±	6.4	42.7 ^{c,§}	±	6.2				TG = 0.74
	HC-HTG	44.5	±	6.4	43.8 [§]	±	6.1				D x TG = 0.74
	HP	46.3	±	7.7	45.5 [§]	±	7.4	45.6 [†]	±	0.4	T x D = 0.46
	HC	43.6	±	6.4	43.0 ^{a,§}	±	6.2	43.5	±	0.4	T x TG = 0.71
	LTG	45.2	±	7.6	44.5 [§]	±	7.2	44.7	±	0.3	T x D x TG = 0.12
	HTG	44.9	±	6.7	44.2 [§]	±	6.5	44.5	±	0.5	
	Time	45.1	±	7.3	44.4 [*]	±	7.0				
Body Fat (%)	HP-LTG	45.6	±	4.4	44.0 [§]	±	4.7				T = 0.001
	HP-HTG	45.4	±	4.5	43.8 [§]	±	4.7				D = 0.036
	HC-LTG	44.9	±	4.3	43.5 [§]	±	4.4				TG = 0.27
	HC-HTG	44.3	±	3.7	43.0 [§]	±	4.0				D x TG = 0.27
	HP	45.5	±	4.4	43.9 [§]	±	4.7	44.7 [†]	±	0.2	T x D = 0.14
	HC	44.7	±	4.2	43.4 [§]	±	4.3	43.9	±	0.3	T x TG = 0.90
	LTG	45.3	±	4.4	43.8 [§]	±	4.6	44.5	±	0.2	T x D x TG = 0.74
	HTG	44.9	±	4.2	43.4 [§]	±	4.4	44.1	±	0.3	
	Time	45.2	±	4.3	43.7 [*]	±	4.5				
HP = higher protein, HC = higher carbohydrate, LTG = low triglyceride, HTG = high triglyceride, T = time effect, D = diet effect, TG = triglyceride risk factor effect, D x TG = diet by triglyceride risk factor effect, T x D = time by diet effect, T x TG = time by triglyceride risk factor effect, T x D x TG = time by diet by triglyceride risk factor effect. Values are means ± standard deviations (except group means are means ± standard error) from 247 participants in the HP-LTG group, 124 in the HP-HTG group, 196 in the HC-LTG group, 96 in the HC-TG group, 371 in the HP total group, 292 in the HC total group, 443 in the LTG group, 220 in the HTG group, and 663 participants total. * Significantly different than baseline, p < 0.05 (univariate). † Significant diet effect, p < 0.05 (univariate). ‡ Significant triglyceride effect, p < 0.05 (univariate). § Significantly different than HP group, p < 0.05 (post hoc LSD). ¶ Significantly different than LTG group, p < 0.05 (post hoc LSD). ¤ Significantly different than HP-LTG group, p < 0.05 (post hoc LSD). ¨ Significantly different than HP-HTG group, p < 0.05 (post hoc LSD). ˆ Significantly different than HC-LTG group, p < 0.05 (post hoc LSD). * Significantly different than baseline, p < 0.05 (post hoc LSD).											

Table B.2: Body Composition Before and After Ten Weeks of Exercise and Dietary Intervention Based on Triglyceride Status and Measured via Anthropometric Measurements

Variable	Group	Baseline	10 Weeks	Group (SEM)	P-level
Weight (kg)	HP-LTG	97.1 ± 19.6	92.7 ^g ± 18.8		T = 0.001
	HP-HTG	93.1 ± 17.6	89.1 ^{c-g} ± 17.2		D = 0.001
	HC-LTG	88.2 ± 15.4	85.1 ^{c-g} ± 14.7		TG = 0.42
	HC-HTG	89.9 ± 13.8	86.4 ^g ± 13.1		D x TG = 0.058
	HP	95.8 ± 19.1	91.5 ^g ± 18.3	93.0 [†] ± 0.9	T x D = 0.002
	HC	88.8 ± 14.9	85.5 ^{a-g} ± 14.2	87.4 ± 1.1	T x TG = 0.72
	LTG	93.2 ± 18.4	89.3 ^g ± 17.5	90.8 ± 0.8	T x D x TG = 0.14
	HTG	91.7 ± 16.1	88.0 ^g ± 15.6	89.6 ± 1.1	
	Time	92.7 ± 17.7	88.9* ± 16.9		
Body Mass Index (kg/m ²)	HP-LTG	36.2 ± 6.8	34.5 ^g ± 6.6		T = 0.001
	HP-HTG	35.5 ± 6.2	34.0 ^g ± 6.0		D = 0.001
	HC-LTG	33.1 ± 5.5	32.0 ^{c-g} ± 5.2		TG = 0.83
	HC-HTG	33.6 ± 4.7	32.3 ^{d-g} ± 4.4		D x TG = 0.29
	HP	35.9 ± 6.6	34.3 ^g ± 6.4	35.0 [†] ± 0.3	T x D = 0.002
	HC	33.3 ± 5.2	32.1 ^{a-g} ± 5.0	32.8 ± 0.4	T x TG = 0.85
	LTG	34.8 ± 6.4	33.4 ^g ± 6.1	34.0 ± 0.3	T x D x TG = 0.24
	HTG	34.7 ± 5.6	33.3 ^g ± 5.4	33.8 ± 0.4	
	Time	34.8 ± 6.2	33.3* ± 5.9		
Waist Circumference (cm)	HP-LTG	101.2 ± 13.8	97.5 ^g ± 13.7		T = 0.001
	HP-HTG	101.6 ± 12.8	97.0 ^g ± 12.1		D = 0.001
	HC-LTG	95.2 ± 11.8	91.7 ^{c-g} ± 11.0		TG = 0.28
	HC-HTG	96.9 ± 11.4	94.5 ^g ± 11.4		D x TG = 0.26
	HP	101.3 ± 13.5	97.3 ^g ± 13.2	99.3 [†] ± 0.7	T x D = 0.015
	HC	95.8 ± 11.7	92.6 ^{a-g} ± 11.2	94.6 ± 0.8	T x TG = 0.79
	LTG	98.6 ± 13.3	94.9 ^g ± 12.9	96.4 ± 0.6	T x D x TG = 0.035
	HTG	99.6 ± 12.4	95.9 ^g ± 11.8	97.5 ± 0.8	
	Time	98.9 ± 13.0	95.2* ± 12.6		
Hip Circumference (cm)	HP-LTG	123.1 ± 13.9	119.9 ^g ± 13.6		T = 0.001
	HP-HTG	122.0 ± 13.8	118.9 ^g ± 13.8		D = 0.001
	HC-LTG	117.8 ± 12.0	115.5 ^{c-g} ± 10.9		TG = 0.97
	HC-HTG	119.4 ± 10.5	115.9 ^g ± 10.8		D x TG = 0.34
	HP	122.7 ± 13.9	119.5 ^g ± 13.6	121.0 [†] ± 0.7	T x D = 0.48
	HC	118.3 ± 11.5	115.6 ^{a-g} ± 10.9	117.2 ± 0.8	T x TG = 0.22
	LTG	120.8 ± 13.3	117.9 ^g ± 12.7	119.1 ± 0.6	T x D x TG = 0.13
	HTG	120.9 ± 12.5	117.6 ^g ± 12.6	119.0 ± 0.8	
	Time	120.8 ± 13.1	117.8* ± 12.6		
HP = higher protein, HC = higher carbohydrate, LTG = low triglyceride, HTG = high triglyceride, T = time effect, D = diet effect, TG = triglyceride risk factor effect, D x TG = diet by triglyceride risk factor effect, T x D = time by diet effect, T x TG = time by triglyceride risk factor effect, T x D x TG = time by diet by triglyceride risk factor effect. Values are means ± standard deviations (except group means are means ± standard error) from 247 participants in the HP-LTG group, 124 in the HP-HTG group, 196 in the HC-LTG group, 96 in the HC-TG group, 371 in the HP total group, 292 in the HC total group, 443 in the LTG group, 220 in the HTG group, and 663 participants total. * Significantly different than baseline, p < 0.05 (univariate). † Significant diet effect, p < 0.05 (univariate). ‡ Significant triglyceride effect, p < 0.05 (univariate). ^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than LTG group, p < 0.05 (post hoc LSD). ^c Significantly different than HP-LTG group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-HTG group, p < 0.05 (post hoc LSD). ^e Significantly different than HC-LTG group, p < 0.05 (post hoc LSD). ^f Significantly different than baseline, p < 0.05 (post hoc LSD).					

Table B.3: Resting Energy Expenditure After Ten Weeks of Exercise and Dietary Intervention Based on Triglyceride Status

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Resting Energy Expenditure (kcal/day)	HP-LTG	1689.6	± 294.2	1628.9 [§]	± 291.9				T = 0.001
	HP-HTG	1713.2	± 266.7	1643.8 [§]	± 294.6				D = 0.001
	HC-LTG	1584.2	± 241.0	1542.4 ^{c,§}	± 247.9				TG = 0.13
	HC-HTG	1635.8	± 260.2	1583.5 [§]	± 249.9				D x TG = 0.53
	HP	1697.4	± 285.2	1633.8 [§]	± 292.5	1668.9 [†]	± 14.1		T x D = 0.32
	HC	1600.7	± 248.0	1555.5 ^{a,§}	± 248.8	1586.5	± 16.2		T x TG = 0.60
	LTG	1643.2	± 276.8	1590.8 [§]	± 276.4	1611.2	± 12.2		T x D x TG = 0.96
	HTG	1680.2	± 266.1	1618.1 [§]	± 277.4	1644.1	± 17.6		
	Time	1655.2	± 273.7	1599.7 [*]	± 276.8				

HP = higher protein, HC = higher carbohydrate, LTG = low triglyceride, HTG = high triglyceride, T = time effect, D = diet effect, TG = triglyceride risk factor effect, D x TG = diet by triglyceride risk factor effect, T x D = time by diet effect, T x TG = time by triglyceride risk factor effect, T x D x TG = time by diet by triglyceride risk factor effect.

Values are means ± standard deviations (except group means are means ± standard error) from 239 participants in the HP-LTG group, 118 in the HP-HTG group, 188 in the HC-LTG group, 88 in the HC-TG group, 357 in the HP total group, 276 in the HC total group, 427 in the LTG group, 206 in the HTG group, and 633 participants total.

* Significantly different than baseline, $p < 0.05$ (univariate). [†] Significant diet effect, $p < 0.05$ (univariate). [‡] Significant triglyceride effect, $p < 0.05$ (univariate). ^a Significantly different than HP group, $p < 0.05$ (post hoc LSD). ^b Significantly different than LTG group, $p < 0.05$ (post hoc LSD). ^c Significantly different than HP-LTG group, $p < 0.05$ (post hoc LSD). ^d Significantly different than HP-HTG group, $p < 0.05$ (post hoc LSD). ^e Significantly different than HC-LTG group, $p < 0.05$ (post hoc LSD). [§] Significantly different than baseline, $p < 0.05$ (post hoc LSD).

Table B.4: Resting Hemodynamic Measurements After Ten Weeks of Exercise and Dietary Intervention Based on Triglyceride Status

Variable	Group	Baseline			10 Weeks			Group (SEM)			P-level
Resting Heart Rate (bpm)	HP-LTG	71.6	±	10.0	68.6 ^a	±	10.1				T = 0.001
	HP-HTG	72.8	±	10.9	69.5 ^b	±	8.8				D = 0.42
	HC-LTG	70.4	±	9.8	67.7 ^b	±	10.2				TG = 0.035
	HC-HTG	72.8	±	11.8	69.3 ^{d,g}	±	9.3				D x TG = 0.51
	HP	72.0	±	10.3	68.9 ^b	±	9.7	70.6	±	0.5	T x D = 0.95
	HC	71.2	±	10.5	68.2 ^b	±	9.9	70.0	±	0.5	T x TG = 0.53
	LTG	71.1	±	9.9	68.2 ^b	±	10.2	69.6	±	0.4	T x D x TG = 0.67
	HTG	72.8	±	11.3	69.4 ^b	±	9.0	71.1 [‡]	±	0.6	
	Time	71.6	±	10.4	68.6 [*]	±	9.8				
Resting Systolic Blood Pressure (mmHg)	HP-LTG	68.6	±	10.1	124.8 ^b	±	14.6				T = 0.001
	HP-HTG	69.5	±	8.8	127.0 ^b	±	14.9				D = 0.07
	HC-LTG	67.7	±	10.2	123.8	±	14.7				TG = 0.60
	HC-HTG	69.3	±	9.3	125.3 ^{d,g}	±	13.8				D x TG = 0.35
	HP	68.9	±	9.7	125.6 ^b	±	14.7	124.5 [†]	±	0.7	T x D = 0.46
	HC	68.2	±	9.9	124.3 ^{a,g}	±	14.4	122.7	±	0.8	T x TG = 0.036
	LTG	68.2	±	10.2	124.4 ^b	±	14.6	123.3	±	0.6	T x D x TG = 0.35
	HTG	69.4	±	9.0	126.3 ^b	±	14.4	123.9	±	0.8	
	Time	68.6	±	9.8	125.0 [*]	±	14.6				
Resting Diastolic Blood Pressure (mmHg)	HP-LTG	124.8	±	14.6	122.7 ^b	±	14.3				T = 0.001
	HP-HTG	127.0	±	14.9	123.5	±	12.5				D = 0.001
	HC-LTG	123.8	±	14.7	121.9 ^b	±	14.2				TG = 0.65
	HC-HTG	125.3	±	13.8	119.7 ^{d,g}	±	14.0				D x TG = 0.96
	HP	125.6	±	14.7	123.0 ^b	±	13.7	80.4 [†]	±	0.4	T x D = 0.25
	HC	124.3	±	14.4	121.2 ^{a,g}	±	14.1	78.1	±	0.5	T x TG = 0.31
	LTG	124.4	±	14.6	122.4 ^b	±	14.2	79.4	±	0.4	T x D x TG = 0.27
	HTG	126.3	±	14.4	121.8 ^b	±	13.3	79.1	±	0.5	
	Time	125.0	±	14.6	122.2 [*]	±	13.9				

HP = higher protein, HC = higher carbohydrate, LTG = low triglyceride, HTG = high triglyceride, T = time effect, D = diet effect, TG = triglyceride risk factor effect, D x TG = diet by triglyceride risk factor effect, T x D = time by diet effect, T x TG = time by triglyceride risk factor effect, T x D x TG = time by diet by triglyceride risk factor effect.

Values are means ± standard deviations (except group means are means ± standard error) from 247 participants in the HP-LTG group, 124 in the HP-HTG group, 196 in the HC-LTG group, 96 in the HC-TG group, 371 in the HP total group, 292 in the HC total group, 443 in the LTG group, 220 in the HTG group, and 663 participants total.

* Significantly different than baseline, p < 0.05 (univariate). † Significant diet effect, p < 0.05 (univariate). ‡ Significant triglyceride effect, p < 0.05 (univariate). ^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than LTG group, p < 0.05 (post hoc LSD). ^c Significantly different than HP-LTG group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-HTG group, p < 0.05 (post hoc LSD). ^e Significantly different than HC-LTG group, p < 0.05 (post hoc LSD). ^f Significantly different than baseline, p < 0.05 (post hoc LSD).

Table B.5: Fasting Blood Lipid Values After Ten Weeks of Exercise and Dietary Intervention Based on Triglyceride Status

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Total Cholesterol (mmol/L)	HP-LTG	5.0	± 0.8	4.9 ^g	± 0.9				T = 0.001
	HP-HTG	5.5	± 1.1	5.2 ^{c,g}	± 1.0				D = 0.61
	HC-LTG	5.0	± 1.0	4.9 ^g	± 0.9				TG = 0.001
	HC-HTG	5.5	± 1.1	5.4 ^f	± 1.0				D x TG = 0.76
	HP	5.2	± 1.0	5.0 ^g	± 1.0	5.2	± 0.05		T x D = 0.24
	HC	5.2	± 1.0	5.0 ^g	± 1.0	5.2	± 0.05		T x TG = 0.45
	LTG	5.0	± 0.9	4.9 ^{b,g}	± 0.9	5.0	± 0.04		T x D x TG = 0.19
	HTG	5.5	± 1.1	5.3 ^g	± 1.0	5.4 [‡]	± 0.06		
	Time	5.2	± 1.0	5.0 [*]	± 1.0				
LDL (mmol/L)	HP-LTG	3.0	± 0.7	2.9 ^g	± 0.7				T = 0.001
	HP-HTG	3.3	± 0.9	3.1 ^{c,g}	± 0.8				D = 0.33
	HC-LTG	3.0	± 0.8	2.9 ^g	± 0.8				TG = 0.001
	HC-HTG	3.3	± 0.9	3.3 ^f	± 0.8				D x TG = 0.33
	HP	3.1	± 0.8	3.0 ^g	± 0.8	3.1	± 0.04		T x D = 0.31
	HC	3.1	± 0.8	3.0 ^g	± 0.8	3.1	± 0.04		T x TG = 0.68
	LTG	3.0	± 0.7	2.9 ^{b,g}	± 0.7	3.0	± 0.03		T x D x TG = 0.08
	HTG	3.3	± 0.9	3.2 ^g	± 0.8	3.2 [‡]	± 0.05		
	Time	3.1	± 0.8	3.0 [*]	± 0.8				
HDL (mmol/L)	HP-LTG	1.4	± 0.3	1.4 ^g	± 0.3				T = 0.001
	HP-HTG	1.3	± 0.3	1.2 ^{c,g}	± 0.3				D = 0.83
	HC-LTG	1.4	± 0.3	1.4 ^g	± 0.3				TG = 0.001
	HC-HTG	1.3	± 0.3	1.2 ^f	± 0.3				D x TG = 0.81
	HP	1.4	± 0.3	1.3 ^g	± 0.3	1.3	± 0.02		T x D = 0.60
	HC	1.4	± 0.3	1.3 ^g	± 0.3	1.3	± 0.02		T x TG = 0.094
	LTG	1.4	± 0.3	1.4 ^{b,g}	± 0.3	1.4	± 0.01		T x D x TG = 0.52
	HTG	1.3	± 0.3	1.2 ^g	± 0.3	1.3 [‡]	± 0.02		
	Time	1.4	± 0.3	1.3 [*]	± 0.3				
Triglycerides (mmol/L)	HP-LTG	1.2	± 0.4	1.2	± 0.5				T = 0.001
	HP-HTG	2.5	± 0.9	2.1 ^{c,g}	± 1.0				D = 0.28
	HC-LTG	1.1	± 0.4	1.2 ^g	± 0.5				TG = 0.001
	HC-HTG	2.3	± 0.6	2.0 ^{f,g}	± 0.8				D x TG = 0.35
	HP	1.6	± 0.8	1.5 ^g	± 0.8	1.7	± 0.03		T x D = 0.051
	HC	1.5	± 0.7	1.5 ^g	± 0.7	1.7	± 0.03		T x TG = 0.001
	LTG	1.1	± 0.4	1.2 ^b	± 0.5	1.2	± 0.03		T x D x TG = 0.90
	HTG	2.4	± 0.8	2.0 ^g	± 0.9	2.2 [‡]	± 0.04		
	Time	1.6	± 0.8	1.5 [*]	± 0.8				
TC/HDL ratio	HP-LTG	3.7	± 0.8	3.8 ^g	± 0.8				T = 0.11
	HP-HTG	4.4	± 0.9	4.3 ^c	± 1.0				D = 0.78
	HC-LTG	3.6	± 0.9	3.7	± 0.9				TG = 0.001
	HC-HTG	4.4	± 1.0	4.5 ^f	± 1.1				D x TG = 0.47
	HP	3.9	± 0.9	3.9	± 0.9	4.0	± 0.05		T x D = 0.23
	HC	3.9	± 1.0	4.0	± 1.1	4.1	± 0.05		T x TG = 0.16
	LTG	3.6	± 0.8	3.7 ^{b,g}	± 0.9	3.7	± 0.04		T x D x TG = 0.12
	HTG	4.4	± 1.0	4.4	± 1.0	4.4 [‡]	± 0.06		
	Time	3.9	± 0.9	4.0	± 1.0				

HP = higher protein, HC = higher carbohydrate, LTG = low triglyceride, HTG = high triglyceride, T = time effect, D = diet effect, TG = triglyceride risk factor effect, D x TG = diet by triglyceride risk factor effect, T x D = time by diet effect, T x TG = time by triglyceride risk factor effect, T x D x TG = time by diet by triglyceride risk factor effect.

Values are means ± standard deviations (except group means are means ± standard error) from 247 participants in the HP-LTG group, 124 in the HP-HTG group, 196 in the HC-LTG group, 96 in the HC-TG group, 371 in the HP total group, 292 in the HC total group, 443 in the LTG group, 220 in the HTG group, and 663 participants total.

* Significantly different than baseline, $p < 0.05$ (univariate). † Significant diet effect, $p < 0.05$ (univariate). ‡ Significant triglyceride effect, $p < 0.05$ (univariate). ^a Significantly different than HP group, $p < 0.05$ (post hoc LSD). ^b Significantly different than LTG group, $p < 0.05$ (post hoc LSD). ^c Significantly different than HP-LTG group, $p < 0.05$ (post hoc LSD). ^d Significantly different than HP-HTG group, $p < 0.05$ (post hoc LSD). ^e Significantly different than HC-LTG group, $p < 0.05$ (post hoc LSD). ^f Significantly different than baseline, $p < 0.05$ (post hoc LSD).

Table B.6: Fasting Glucose and Insulin Values After Ten Weeks of Exercise and Dietary Intervention Based on Triglyceride Status

Variable	Group	Baseline			10 Weeks			Group (SEM)			P-level
Glucose (mmol/L)	HP-LTG	99.6	±	18.3	98.1	±	16.7				T = 0.006
	HP-HTG	109.4	±	27.7	105.9 ^{c,g}	±	28.0				D = 0.29
	HC-LTG	98.3	±	15.9	97.6	±	13.6				TG = 0.001
	HC-HTG	106.4	±	33.7	104.0 ^f	±	25.0				D x TG = 0.63
	HP	102.9	±	22.4	100.7 ^e	±	21.4	103.3	±	1.07	T x D = 0.50
	HC	100.9	±	23.6	99.7	±	18.4	101.6	±	1.21	T x TG = 0.21
	LTG	99.0	±	17.3	97.9 ^b	±	15.4	98.4	±	0.93	T x D x TG = 0.92
	HTG	108.1	±	30.4	105.1 ^g	±	26.7	106.4 [‡]	±	1.32	
Time	102.0	±	22.9	100.3 [*]	±	20.1					
Insulin (uIU/mL)	HP-LTG	3.1	±	8.3	3.5	±	9.7				T = 0.66
	HP-HTG	5.8	±	9.5	6.4	±	10.7				D = 0.002
	HC-LTG	6.9	±	12.1	7.7 ^c	±	11.5				TG = 0.031
	HC-HTG	10.5	±	13.9	9.8	±	12.9				D x TG = 0.98
	HP	4.0	±	8.8	4.5	±	10.1	4.7 [†]	±	0.84	T x D = 0.67
	HC	8.3	±	12.8	8.4 ^a	±	12.0	8.7	±	0.99	T x TG = 0.62
	LTG	4.6	±	10.1	5.2	±	10.6	5.3	±	0.77	T x D x TG = 0.53
	HTG	7.8	±	11.7	7.9	±	11.7	8.1 [‡]	±	1.04	
Time	5.7	±	10.8	6.1	±	11.1					
Calculated HOMA	HP-LTG	0.8	±	2.1	0.9	±	2.4				T = 0.98
	HP-HTG	1.6	±	2.6	1.9	±	3.6				D = 0.003
	HC-LTG	1.8	±	3.3	2.0 ^c	±	3.5				TG = 0.010
	HC-HTG	3.2	±	6.2	2.6	±	3.5				D x TG = 0.91
	HP	1.0	±	2.3	1.2	±	2.9	1.3 [†]	±	0.24	T x D = 0.42
	HC	2.3	±	4.6	2.2 ^a	±	3.5	2.4	±	0.29	T x TG = 0.40
	LTG	1.2	±	2.7	1.3	±	3.0	1.4	±	0.22	T x D x TG = 0.22
	HTG	2.3	±	4.6	2.2	±	3.6	2.3 [‡]	±	0.30	
Time	1.6	±	3.5	1.6	±	3.2					
HP = higher protein, HC = higher carbohydrate, LTG = low triglyceride, HTG = high triglyceride, T = time effect, D = diet effect, TG = triglyceride risk factor effect, D x TG = diet by triglyceride risk factor effect, T x D = time by diet effect, T x TG = time by triglyceride risk factor effect, T x D x TG = time by diet by triglyceride risk factor effect. Glucose values are means ± standard deviations (except group means are means ± standard error) from 247 participants in the HP-LTG group, 124 in the HP-HTG group, 196 in the HC-LTG group, 96 in the HC-TG group, 371 in the HP total group, 292 in the HC total group, 443 in the LTG group, 220 in the HTG group, and 663 participants total. Insulin and HOMA values are means ± standard deviations (except group means are means ± standard error) from 99 participants in the HP-LTG group, 50 in the HP-HTG group, 65 in the HC-LWC group, 38 in the HC-HWC group, 149 in the HP total group, 103 in the HC total group, 164 in the LWC group, 88 in the HWC group, and 252 participants total. [*] Significantly different than baseline, p < 0.05 (univariate). [†] Significant diet effect, p < 0.05 (univariate). [‡] Significant triglyceride effect, p < 0.05 (univariate). ^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than LTG group, p < 0.05 (post hoc LSD). ^c Significantly different than HP-LTG group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-HTG group, p < 0.05 (post hoc LSD). ^e Significantly different than HC-LTG group, p < 0.05 (post hoc LSD). ^f Significantly different than baseline, p < 0.05 (post hoc LSD).											

Table B.7: Fitness Parameters After Ten Weeks of Exercise and Dietary Intervention Based on Triglyceride Status

Variable	Group	Baseline			10 Weeks			Group (SEM)			P-level
Bench Press Max Strength (1 RM / kg body weight)	HP-LTG	31.5	±	7.9	33.7 ^g	±	7.6				T = 0.001
	HP-HTG	29.7	±	6.8	31.3 ^{c,g}	±	6.7				D = 0.76
	HC-LTG	29.3	±	7.7	31.8 ^{c,g}	±	8.0				TG = 0.69
	HC-HTG	31.2	±	8.0	33.1 ^g	±	8.3				D x TG = 0.004
	HP	30.9	±	7.6	32.9 ^g	±	7.4	31.6	±	0.4	T x D = 0.41
	HC	29.9	±	7.8	32.2 ^g	±	8.1	31.4	±	0.5	T x TG = 0.18
	LTG	30.6	±	7.9	32.9 ^g	±	7.8	31.6	±	0.4	T x D x TG = 0.96
	HTG	30.3	±	7.3	32.1 ^g	±	7.4	31.3	±	0.5	
	Time	30.5	±	7.7	32.6 [*]	±	7.7				
Bench Press Lift Volume (kg)	HP-LTG	181.7	±	76.3	186.2	±	83.5				T = 0.89
	HP-HTG	177.3	±	86.8	173.2	±	74.1				D = 0.46
	HC-LTG	182.4	±	83.3	174.7	±	77.1				TG = 0.84
	HC-HTG	187.0	±	87.6	192.3	±	85.1				D x TG = 0.10
	HP	180.3	±	79.7	182.0	±	80.7	179.6	±	3.9	T x D = 0.86
	HC	183.8	±	84.5	180.1	±	79.9	184.1	±	4.6	T x TG = 0.78
	LTG	182.0	±	79.3	181.2	±	80.9	181.3	±	3.4	T x D x TG = 0.17
	HTG	181.4	±	87.0	181.2	±	79.2	182.5	±	5.0	
	Time	181.8	±	81.8	181.2	±	80.3				
Leg Press Max Strength (1 RM / kg body weight)	HP-LTG	166.4	±	47.1	181.3 ^g	±	50.1				T = 0.001
	HP-HTG	159.3	±	50.0	177.6 ^g	±	56.7				D = 0.45
	HC-LTG	155.1	±	51.1	168.2 ^{c,g}	±	55.5				TG = 0.43
	HC-HTG	165.6	±	49.8	182.3 ^g	±	53.5				D x TG = 0.045
	HP	164.1	±	48.1	180.1 ^g	±	52.3	171.2	±	2.9	T x D = 0.51
	HC	158.4	±	50.8	172.5 ^g	±	55.2	167.8	±	3.3	T x TG = 0.17
	LTG	161.5	±	49.1	175.6 ^g	±	52.9	167.8	±	2.5	T x D x TG = 0.95
	HTG	161.9	±	49.8	179.6 ^g	±	55.3	171.2	±	3.6	
	Time	161.7	±	49.3	176.8 [*]	±	53.6				
Leg Press Lift Volume (kg)	HP-LTG	1796.3	±	899.7	1943.7 ^g	±	918.4				T = 0.40
	HP-HTG	1874.9	±	1100.6	1910.9	±	1356.7				D = 0.016
	HC-LTG	1599.7	±	877.6	1586.6 ^c	±	837.1				TG = 0.13
	HC-HTG	1818.7	±	818.8	1783.6	±	1024.7				D x TG = 0.23
	HP	1821.8	±	968.3	1933.1	±	1077.9	1881.5 [†]	±	49.6	T x D = 0.15
	HC	1667.3	±	864.3	1647.4 ^a	±	901.7	1697.1	±	57.9	T x TG = 0.40
	LTG	1710.8	±	894.4	1788.4	±	900.5	1731.6	±	42.8	T x D x TG = 0.58
	HTG	1851.4	±	990.6	1857.7	±	1227.5	1847.0	±	63.1	
	Time	1755.4	±	927.5	1810.3	±	1015.1				
Peak VO₂ (mL/kg/min)	HP-LTG	20.2	±	4.6	22.1 ^g	±	4.6				T = 0.001
	HP-HTG	19.7	±	4.4	21.7 ^g	±	4.4				D = 0.009
	HC-LTG	20.5	±	4.3	22.9 ^{c,g}	±	4.6				TG = 0.85
	HC-HTG	20.5	±	4.0	23.4 ^{d,g}	±	5.0				D x TG = 0.32
	HP	20.0	±	4.5	21.9 ^g	±	4.5	20.9 [†]	±	0.23	T x D = 0.012
	HC	20.5	±	4.2	23.1 ^{a,g}	±	4.7	21.8	±	0.26	T x TG = 0.30
	LTG	20.3	±	4.5	22.4 ^g	±	4.6	21.4	±	0.20	T x D x TG = 0.67
	HTG	20.1	±	4.2	22.5 ^g	±	4.7	21.3	±	0.28	
	Time	20.2	±	4.4	22.4 [*]	±	4.6				

HP = higher protein, HC = higher carbohydrate, LTG = low triglyceride, HTG = high triglyceride, T = time effect, D = diet effect, TG = triglyceride risk factor effect, D x TG = diet by triglyceride risk factor effect, T x D = time by diet effect, T x TG = time by triglyceride risk factor effect, T x D x TG = time by diet by triglyceride risk factor effect.

Strength values are means ± standard deviations (except group means are means ± standard error) from 230 participants in the HP-LTG group, 110 in the HP-HTG group, 177 in the HC-LTG group, 79 in the HC-TG group, 340 in the HP total group, 256 in the HC total group, 407 in the LTG group, 189 in the HTG group, and 596 participants total. Peak VO₂ values are means ± standard deviations (except group means are means ± standard error) from 247 participants in the HP-LTG group, 124 in the HP-HTG group, 196 in the HC-LTG group, 96 in the HC-TG group, 371 in the HP total group, 292 in the HC total group, 443 in the LTG group, 220 in the HTG group, and 663 participants total.

* Significantly different than baseline, p < 0.05 (univariate). † Significant diet effect, p < 0.05 (univariate). ‡ Significant triglyceride effect, p < 0.05 (univariate). ^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than LTG group, p < 0.05 (post hoc LSD). ^c Significantly different than HP-LTG group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-HTG group, p < 0.05 (post hoc LSD). ^f Significantly different than HC-LTG group, p < 0.05 (post hoc LSD). ^g Significantly different than baseline, p < 0.05 (post hoc LSD).

Table B.8: Changes in SF36 - Quality of Life Inventory Values After Ten Weeks of Exercise and Dietary Intervention Based on Triglyceride Status

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Physical Functioning	HP-LTG	77.1	± 33.3	84.2 ^s	± 27.0				T = 0.001
	HP-HTG	72.9	± 21.4	75.8 ^c	± 18.6				D = 0.43
	HC-LTG	75.8	± 24.5	83.3 ^s	± 21.3				TG = 0.23
	HC-HTG	77.2	± 14.8	82.0	± 18.5				D x TG = 0.24
	HP	75.7	± 29.7	81.3 ^s	± 24.7	77.5	± 1.85		T x D = 0.67
	HC	76.4	± 21.2	82.8 ^s	± 20.2	79.6	± 1.90		T x TG = 0.22
	LTG	76.5	± 29.5	83.8 ^s	± 24.5	80.1	± 1.60		T x D x TG = 0.79
	HTG	75.1	± 18.4	78.9	± 18.7	76.9	± 2.11		
	Time	76.0	± 26.0	82.0*	± 22.7				
Role Physical	HP-LTG	149.8	± 140.4	158.5	± 153.8				T = 0.52
	HP-HTG	127.6	± 132.0	123.3	± 134.4				D = 0.001
	HC-LTG	260.1	± 138.9	265.4 ^c	± 143.3				TG = 0.08
	HC-HTG	230.4	± 147.0	231.5 ^d	± 152.0				D x TG = 0.93
	HP	142.2	± 137.5	146.4	± 148.0	139.8†	± 11.91		T x D = 0.91
	HC	248.6	± 142.3	252.3 ^a	± 147.1	246.9	± 12.22		T x TG = 0.31
	LTG	200.1	± 149.8	207.3	± 158.0	208.4	± 10.31		T x D x TG = 0.60
	HTG	179.5	± 148.4	177.9	± 152.9	178.2	± 13.59		
	Time	192.6	± 149.4	196.6	± 156.5				
Bodily Pain	HP-LTG	58.5	± 18.9	63.2 ^s	± 20.4				T = 0.039
	HP-HTG	60.2	± 19.2	62.9	± 21.5				D = 0.42
	HC-LTG	59.2	± 21.9	63.0 ^s	± 20.9				TG = 0.31
	HC-HTG	66.4	± 20.2	63.8	± 22.9				D x TG = 0.47
	HP	59.1	± 19.0	63.1 ^s	± 20.7	61.2	± 1.61		T x D = 0.13
	HC	62.0	± 21.4	63.3	± 21.6	63.1	± 1.65		T x TG = 0.047
	LTG	58.8	± 20.3	63.1 ^s	± 20.6	61.0	± 1.39		T x D x TG = 0.29
	HTG	63.3	± 19.9	63.4	± 22.1	63.3	± 1.84		
	Time	60.5	± 20.2	63.2*	± 21.1				
General Health	HP-LTG	49.5	± 28.9	55.3 ^s	± 28.8				T = 0.001
	HP-HTG	51.9	± 28.2	53.9	± 28.5				D = 0.001
	HC-LTG	66.4	± 21.5	69.6 ^{c,s}	± 21.7				TG = 0.66
	HC-HTG	63.1	± 23.6	66.4 ^{d,s}	± 23.0				D x TG = 0.55
	HP	50.4	± 28.6	54.8 ^s	± 28.6	52.6†	± 2.15		T x D = 0.72
	HC	65.1	± 22.3	68.4 ^{a,s}	± 22.1	66.4	± 2.21		T x TG = 0.21
	LTG	57.2	± 27.1	61.8 ^s	± 26.7	60.2	± 1.86		T x D x TG = 0.18
	HTG	57.6	± 26.5	60.2 ^s	± 26.5	58.8	± 2.46		
	Time	57.3	± 26.8	61.2*	± 26.6				
Vital	HP-LTG	37.2	± 22.3	45.5 ^s	± 27.8				T = 0.001
	HP-HTG	49.0	± 72.3	56.0 ^s	± 71.5				D = 0.18
	HC-LTG	50.7	± 17.3	56.5 ^{c,s}	± 17.1				TG = 0.28
	HC-HTG	49.9	± 20.4	54.0 ^s	± 20.7				D x TG = 0.14
	HP	41.3	± 46.1	49.1 ^s	± 47.5	46.9	± 3.03		T x D = 0.13
	HC	50.4	± 18.5	55.5 ^s	± 18.6	52.8	± 3.11		T x TG = 0.41
	LTG	43.4	± 21.2	50.6 ^s	± 24.1	47.5	± 2.62		T x D x TG = 0.90
	HTG	49.4	± 52.7	55.0 ^s	± 52.2	52.2	± 3.46		
	Time	45.6	± 36.0	52.2*	± 36.8				

Table B.8: Continued

Variable	Group	Baseline	10 Weeks	Group (SEM)	P-level
Social Functioning	HP-LTG	46.0 ± 25.7	49.9 ^a ± 25.8		T = 0.009
	HP-HTG	46.8 ± 26.1	52.3 ^b ± 28.7		D = 0.001
	HC-LTG	61.4 ± 25.1	62.1 ^c ± 24.1		TG = 0.52
	HC-HTG	55.8 ± 24.5	56.9 ± 25.0		D x TG = 0.23
	HP	46.2 ± 25.7	50.7 ^b ± 26.7	48.7 [†] ± 2.05	T x D = 0.07
	HC	59.3 ± 24.9	60.1 ^a ± 24.5	59.1 ± 2.11	T x TG = 0.66
	LTG	53.0 ± 26.5	55.5 ± 25.7	54.8 ± 1.78	T x D x TG = 0.77
	HTG	51.4 ± 25.6	54.6 ^b ± 26.9	52.9 ± 2.35	
	Time	52.4 ± 26.1	55.2* ± 26.1		
Role Emotional	HP-LTG	224.3 ± 128.5	241.4 ± 140.6		T = 0.33
	HP-HTG	206.6 ± 129.6	233.1 ± 141.8		D = 0.001
	HC-LTG	312.5 ± 219.9	290.2 ^c ± 131.4		TG = 0.33
	HC-HTG	276.4 ± 129.5	288.0 ^d ± 139.1		D x TG = 0.85
	HP	218.3 ± 128.7	238.6 ± 140.6	226.4 [†] ± 11.38	T x D = 0.11
	HC	298.6 ± 190.3	289.4 ^a ± 133.9	291.8 ± 11.68	T x TG = 0.20
	LTG	264.6 ± 181.1	263.7 ± 138.3	267.1 ± 9.85	T x D x TG = 0.47
	HTG	241.8 ± 133.6	260.9 ± 142.4	251.1 ± 13.00	
	Time	256.3 ± 165.6	262.7 ± 139.6		
Mental Health	HP-LTG	56.9 ± 15.4	65.5 ^b ± 15.6		T = 0.001
	HP-HTG	55.9 ± 15.9	64.5 ^b ± 14.9		D = 0.019
	HC-LTG	60.2 ± 14.2	65.6 ^b ± 11.7		TG = 0.58
	HC-HTG	60.4 ± 14.7	70.8 ^{d,f,g} ± 12.2		D x TG = 0.22
	HP	56.5 ± 15.5	65.1 ^b ± 15.3	60.7 [†] ± 1.06	T x D = 0.71
	HC	60.3 ± 14.4	67.6 ^b ± 12.1	64.2 ± 1.08	T x TG = 0.17
	LTG	58.4 ± 14.9	65.5 ^b ± 13.9	62.0 ± 0.92	T x D x TG = 0.18
	HTG	58.1 ± 15.4	67.7 ^b ± 13.9	62.9 ± 1.21	
	Time	58.3 ± 15.1	66.3* ± 13.9		
<p>HP = higher protein, HC = higher carbohydrate, LTG = low triglyceride, HTG = high triglyceride, T = time effect, D = diet effect, TG = triglyceride risk factor effect, D x TG = diet by triglyceride risk factor effect, T x D = time by diet effect, T x TG = time by triglyceride risk factor effect, T x D x TG = time by diet by triglyceride risk factor effect.</p> <p>Values are means ± standard deviations (except group means are means ± standard error) from 100 participants in the HP-LTG group, 52 in the HP-HTG group, 84 in the HC-LTG group, 53 in the HC-TG group, 152 in the HP total group, 137 in the HC total group, 184 in the LTG group, 105 in the HTG group, and 289 participants total.</p> <p>* Significantly different than baseline, $p < 0.05$ (univariate). † Significant diet effect, $p < 0.05$ (univariate). ‡ Significant triglyceride effect, $p < 0.05$ (univariate). ^a Significantly different than HP group, $p < 0.05$ (post hoc LSD). ^b Significantly different than LTG group, $p < 0.05$ (post hoc LSD). ^c Significantly different than HP-LTG group, $p < 0.05$ (post hoc LSD). ^d Significantly different than HP-HTG group, $p < 0.05$ (post hoc LSD). ^e Significantly different than HC-LTG group, $p < 0.05$ (post hoc LSD). ^f Significantly different than baseline, $p < 0.05$ (post hoc LSD).</p>					

Table B.9: Changes in Body Image Evaluation After Ten Weeks of Exercise and Dietary Intervention Based on Triglyceride Status

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Appearance Evaluation	HP-LTG	2.4	± 0.6	2.8 ^g	± 0.7				T = 0.001
	HP-HTG	2.4	± 0.6	2.7 ^g	± 0.6				D = 0.55
	HC-LTG	2.5	± 0.6	2.8 ^g	± 0.7				TG = 0.26
	HC-HTG	2.4	± 0.6	2.8 ^g	± 0.7				D x TG = 0.62
	HP	2.4	± 0.6	2.7 ^g	± 0.7	2.6	± 0.04		T x D = 0.58
	HC	2.4	± 0.6	2.8 ^g	± 0.7	2.6	± 0.04		T x TG = 0.71
	LTG	2.4	± 0.6	2.8 ^g	± 0.7	2.6	± 0.03		T x D x TG = 0.96
	HTG	2.4	± 0.6	2.7 ^g	± 0.6	2.6	± 0.05		
	Time	2.4	± 0.6	2.8*	± 0.7				
Appearance Orientation	HP-LTG	4.2	± 0.8	4.1	± 0.9				T = 0.49
	HP-HTG	4.1	± 0.9	4.1	± 0.8				D = 0.020
	HC-LTG	4.0	± 0.8	3.9 ^c	± 0.9				TG = 0.41
	HC-HTG	3.9	± 0.8	3.9	± 0.8				D x TG = 0.96
	HP	4.1	± 0.9	4.1	± 0.9	4.1†	± 0.06		T x D = 0.36
	HC	3.9	± 0.8	3.9 ^a	± 0.9	3.9	± 0.06		T x TG = 0.23
	LTG	4.1	± 0.8	4.0	± 0.9	4.1	± 0.05		T x D x TG = 0.77
	HTG	4.0	± 0.8	4.0	± 0.8	4.0	± 0.07		
	Time	4.1	± 0.8	4.0	± 0.9				
Body Area Satisfaction	HP-LTG	2.1	± 0.8	2.3 ^g	± 0.8				T = 0.001
	HP-HTG	2.0	± 0.7	2.3 ^g	± 0.8				D = 0.002
	HC-LTG	2.2	± 0.7	2.6 ^{c,g}	± 0.8				TG = 0.74
	HC-HTG	2.2	± 0.7	2.5 ^g	± 0.8				D x TG = 0.88
	HP	2.0	± 0.8	2.3 ^g	± 0.8	2.2†	± 0.05		T x D = 0.64
	HC	2.2	± 0.7	2.5 ^{a,g}	± 0.8	2.4	± 0.06		T x TG = 0.20
	LTG	2.1	± 0.7	2.4 ^g	± 0.8	2.3	± 0.04		T x D x TG = 0.033
	HTG	2.1	± 0.7	2.4 ^g	± 0.8	2.3	± 0.06		
	Time	2.1	± 0.7	2.4*	± 0.8				
Overweight Preoccupation	HP-LTG	2.9	± 0.7	3.4 ^g	± 0.7				T = 0.001
	HP-HTG	2.9	± 0.6	3.4 ^g	± 0.7				D = 0.29
	HC-LTG	2.9	± 0.7	3.3 ^g	± 0.7				TG = 0.74
	HC-HTG	2.8	± 0.7	3.2 ^g	± 0.7				D x TG = 0.69
	HP	2.9	± 0.7	3.4 ^g	± 0.7	3.1	± 0.04		T x D = 0.12
	HC	2.9	± 0.7	3.3 ^g	± 0.7	3.1	± 0.05		T x TG = 0.98
	LTG	2.9	± 0.7	3.4 ^g	± 0.7	3.1	± 0.04		T x D x TG = 0.62
	HTG	2.9	± 0.7	3.3 ^g	± 0.7	3.1	± 0.05		
	Time	2.9	± 0.7	3.3*	± 0.7				
Self-Classified Weight	HP-LTG	4.3	± 0.7	4.2	± 0.7				T = 0.024
	HP-HTG	4.2	± 0.8	4.2	± 0.6				D = 0.14
	HC-LTG	4.1	± 0.7	4.0	± 0.7				TG = 0.66
	HC-HTG	4.2	± 0.7	4.1	± 0.8				D x TG = 0.39
	HP	4.2	± 0.7	4.2	± 0.7	4.2	± 0.04		T x D = 0.43
	HC	4.2	± 0.7	4.0	± 0.7	4.1	± 0.04		T x TG = 0.87
	LTG	4.2	± 0.7	4.1 ^g	± 0.7	4.1	± 0.03		T x D x TG = 0.48
	HTG	4.2	± 0.8	4.1 ^g	± 0.7	4.2	± 0.05		
	Time	4.2	± 0.7	4.1*	± 0.7				

Table B.9: Continued

Variable	Group	Baseline		10 Weeks			Group (SEM)			P-level
Rosenberg Self Esteem	HP-LTG	26.0	± 4.0	26.6 [§]	± 4.0				T = 0.006	
	HP-HTG	25.1	± 3.7	25.7	± 3.5				D = 0.94	
	HC-LTG	25.8	± 4.0	25.8	± 3.9				TG = 0.28	
	HC-HTG	25.5	± 3.8	26.2	± 4.1				D x TG = 0.20	
	HP	25.7	± 3.9	26.3 [§]	± 3.8	25.8	± 0.24	T x D = 0.38		
	HC	25.7	± 3.9	25.9	± 4.0	25.8	± 0.27	T x TG = 0.37		
	LTG	25.9	± 4.0	26.3	± 4.0	26.0	± 0.21	T x D x TG = 0.32		
	HTG	25.3	± 3.8	25.9 [§]	± 3.8	25.6	± 0.29			
	Time	25.7	± 3.9	26.2*	± 3.9					
Social Physique Anxiety	HP-LTG	31.1	± 6.4	31.2	± 6.5				T = 0.56	
	HP-HTG	31.5	± 6.0	31.8	± 5.6				D = 0.74	
	HC-LTG	31.2	± 6.1	31.0	± 5.1				TG = 0.44	
	HC-HTG	31.0	± 5.7	31.7	± 7.6				D x TG = 0.85	
	HP	31.3	± 6.3	31.4	± 6.2	31.4	± 0.35	T x D = 0.83		
	HC	31.1	± 6.0	31.3	± 6.1	31.2	± 0.39	T x TG = 0.46		
	LTG	31.1	± 6.3	31.1	± 6.0	31.1	± 0.30	T x D x TG = 0.59		
	HTG	31.3	± 5.9	31.7	± 6.6	31.5	± 0.42			
	Time	31.2	± 6.1	31.3	± 6.2					
HP = higher protein, HC = higher carbohydrate, LTG = low triglyceride, HTG = high triglyceride, T = time effect, D = diet effect, TG = triglyceride risk factor effect, D x TG = diet by triglyceride risk factor effect, T x D = time by diet effect, T x TG = time by triglyceride risk factor effect, T x D x TG = time by diet by triglyceride risk factor effect. Values are means ± standard deviations (except group means are means ± standard error) from 177 participants in the HP-LTG group, 79 in the HP-HTG group, 127 in the HC-LTG group, 68 in the HC-TG group, 256 in the HP total group, 195 in the HC total group, 304 in the LTG group, 147 in the HTG group, and 451 participants total. * Significantly different than baseline, p < 0.05 (univariate). † Significant diet effect, p < 0.05 (univariate). ‡ Significant triglyceride effect, p < 0.05 (univariate). ^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than LTG group, p < 0.05 (post hoc LSD). ^c Significantly different than HP-LTG group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-HTG group, p < 0.05 (post hoc LSD). ^e Significantly different than HC-LTG group, p < 0.05 (post hoc LSD). ^f Significantly different than baseline, p <0.05 (post hoc LSD).										

APPENDIX C

HDL CHOLESTEROL RISK FACTOR TABLES

Table C.1: Body Composition Before and After Ten Weeks of Exercise and Dietary Intervention Based on HDL Status and Measured via DEXA Scan

Variable	Group	Baseline			10 Weeks			Group (SEM)			P-level
Scanned Mass (kg)	HP-HHDL	88.9	±	18.4	85.1	±	17.9				T = 0.001
	HP-LHDL	89.7	±	17.7	85.7 [®]	±	16.9				D = 0.001
	HC-HHDL	81.2	±	13.7	78.3 ^c	±	13.1				HDL = 0.14
	HC-LHDL	84.5	±	15.2	81.3 ^d	±	14.5				D x HDL = 0.37
	HP	89.2	±	18.1	85.3 [®]	±	17.5	87.3†	±	0.85	T x D = 0.002
	HC	82.5	±	14.3	79.5 ^{a®}	±	13.7	81.3	±	0.97	T x HDL = 0.35
	HHDL	85.4	±	16.9	82.0 [®]	±	16.2	83.4	±	0.81	T x D x HDL = 0.84
	LHDL	87.5	±	16.8	83.8 [®]	±	16.0	85.3	±	1.01	
Time	86.2	±	16.9	82.7*	±	16.2					
Fat Mass (kg)	HP-HHDL	41.4	±	11.6	38.3	±	11.4				T = 0.001
	HP-LHDL	40.7	±	11.2	37.5 [®]	±	10.9				D = 0.001
	HC-HHDL	36.7	±	9.0	34.4 ^c	±	8.7				HDL = 0.93
	HC-LHDL	37.8	±	9.3	35.1	±	8.7				D x HDL = 0.29
	HP	41.1	±	11.4	38.0 [®]	±	11.2	39.5†	±	0.54	T x D = 0.003
	HC	37.1	±	9.1	34.7 ^{b,c}	±	8.7	36.0	±	0.62	T x HDL = 0.35
	HHDL	39.3	±	10.8	36.6 ^{b,c}	±	10.5	37.7	±	0.51	T x D x HDL = 0.47
	LHDL	39.4	±	10.5	36.5 [®]	±	10.0	37.8	±	0.64	
Time	39.3	±	10.7	36.5*	±	10.3					
Lean Mass (kg)	HP-HHDL	45.6	±	7.8	44.9	±	7.5				T = 0.001
	HP-LHDL	47.3	±	7.5	46.4 ^{a,®}	±	7.1				D = 0.001
	HC-HHDL	42.8	±	6.0	42.2 ^c	±	5.6				HDL = 0.001
	HC-LHDL	44.9	±	6.9	44.3 ^{d,f}	±	6.9				D x HDL = 0.61
	HP	46.3	±	7.7	45.5 [®]	±	7.4	46.0†	±	0.37	T x D = 0.15
	HC	43.6	±	6.4	43.0 ^{a,®}	±	6.2	43.6	±	0.42	T x HDL = 0.80
	HHDL	44.4	±	7.2	43.7 ^{b,®}	±	6.9	43.9	±	0.35	T x D x HDL = 0.45
	LHDL	46.2	±	7.4	45.5 [®]	±	7.1	45.7‡	±	0.43	
Time	45.1	±	7.3	44.4*	±	7.0					
Body Fat (%)	HP-HHDL	46.1	±	4.3	44.5	±	4.6				T = 0.001
	HP-LHDL	44.7	±	4.5	43.1 ^{c,®}	±	4.7				D = 0.06
	HC-HHDL	44.9	±	4.3	43.7	±	4.3				HDL = 0.003
	HC-LHDL	44.3	±	4.0	42.9	±	4.2				D x HDL = 0.33
	HP	45.5	±	4.4	43.9 [®]	±	4.7	44.6†	±	0.23	T x D = 0.21
	HC	44.7	±	4.2	43.4 [®]	±	4.3	44.0	±	0.26	T x HDL = 0.48
	HHDL	45.6	±	4.3	44.1 [®]	±	4.5	44.8	±	0.22	T x D x HDL = 0.46
	LHDL	44.5	±	4.3	43.0 [®]	±	4.5	43.8‡	±	0.27	
Time	45.2	±	4.3	43.7*	±	4.5					
HP = higher protein, HC = higher carbohydrate, HHDL = high HDL cholesterol, LHDL = low HDL cholesterol, T = time effect, D = diet effect, HDL = HDL risk factor effect, D x HDL = diet by HDL risk factor effect, T x D = time by diet effect, T x HDL = time by HDL risk factor effect, T x D x HDL = time by diet by HDL risk factor effect. Values are means ± standard deviations (except group means are means ± standard error) from 220 participants in the HP-HHDL group, 151 in the HP-LHDL group, 180 in the HC-HHDL group, 112 in the HC-LHDL group, 371 in the HP total group, 292 in the HC total group, 400 in the HHDL group, 263 in the LHDL group, and 663 participants total. * Significantly different than baseline, p < 0.05 (univariate). † Significant diet effect, p < 0.05 (univariate). ‡ Significant HDL effect, p < 0.05 (univariate). ^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than HHDL group, p < 0.05 (post hoc LSD). ^c Significantly different than HP-HHDL group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-LHDL group, p < 0.05 (post hoc LSD). ^e Significantly different than HC-HHDL group, p < 0.05 (post hoc LSD). ^f Significantly different than baseline, p < 0.05 (post hoc LSD).											

Table C.2: Body Composition Before and After Ten Weeks of Exercise and Dietary Intervention Based on HDL Status and Measured via Anthropometric Measurements

Variable	Group	Baseline		10 Weeks			Group (SEM)			P-level
Weight (kg)	HP-HHDL	95.4	± 19.4	91.2 [§]	± 18.9				T = 0.001	
	HP-LHDL	96.3	± 18.6	91.9 [§]	± 17.6				D = 0.001	
	HC-HHDL	87.5	± 14.2	84.4 ^{c,§}	± 13.5				HDL = 0.15	
	HC-LHDL	90.8	± 15.8	87.3 ^{d,§}	± 15.2				D x HDL = 0.38	
	HP	95.8	± 19.1	91.5 [§]	± 18.3	93.7 [†]	± 0.9	T x D = 0.001		
	HC	88.8	± 14.9	85.5 ^{a,§}	± 14.2	87.5	± 1.0	T x HDL = 0.40		
	HHDL	91.9	± 17.7	88.1 [§]	± 17.0	89.6	± 0.9	T x D x HDL = 0.77		
	LHDL	93.9	± 17.6	90.0 [§]	± 16.7	91.6 [‡]	± 1.1			
Time	92.7	± 17.7	88.9*	± 16.9						
Body Mass Index (kg/m ²)	HP-HHDL	35.9	± 6.7	34.3 [§]	± 6.5				T = 0.001	
	HP-LHDL	36.0	± 6.4	34.4 [§]	± 6.1				D = 0.001	
	HC-HHDL	32.8	± 5.1	31.6 ^{c,§}	± 4.8				HDL = 0.16	
	HC-LHDL	34.1	± 5.3	32.8 ^{d,§}	± 5.1				D x HDL = 0.19	
	HP	35.9	± 6.6	34.3 [§]	± 6.4	35.1 [†]	± 0.3	T x D = 0.001		
	HC	33.3	± 5.2	32.1 ^{a,§}	± 5.0	32.8	± 0.4	T x HDL = 0.43		
	HHDL	34.5	± 6.2	33.1 [§]	± 6.0	33.7	± 0.3	T x D x HDL = 0.67		
	LHDL	35.2	± 6.0	33.7 [§]	± 5.8	34.3	± 0.4			
Time	34.8	± 6.2	33.3*	± 5.9						
Waist Circumference (cm)	HP-HHDL	101.0	± 14.0	97.0 [§]	± 13.6				T = 0.001	
	HP-LHDL	101.8	± 12.7	97.7 [§]	± 12.6				D = 0.001	
	HC-HHDL	94.6	± 11.3	91.4 ^{c,§}	± 11.0				HDL = 0.055	
	HC-LHDL	97.6	± 12.1	94.5 ^{d,f,§}	± 11.3				D x HDL = 0.24	
	HP	101.3	± 13.5	97.3 [§]	± 13.2	99.4 [†]	± 0.6	T x D = 0.07		
	HC	95.8	± 11.7	92.6 ^{a,§}	± 11.2	94.5	± 0.7	T x HDL = 0.98		
	HHDL	98.2	± 13.2	94.5 [§]	± 12.8	96.0	± 0.6	T x D x HDL = 0.87		
	LHDL	100.0	± 12.6	96.3 [§]	± 12.1	97.9	± 0.8			
Time	98.9	± 13.0	95.2*	± 12.6						
Hip Circumference (cm)	HP-HHDL	123.0	± 14.1	119.9 [§]	± 14.1				T = 0.001	
	HP-LHDL	122.4	± 13.6	119.0 [§]	± 13.0				D = 0.001	
	HC-HHDL	118.0	± 11.4	115.2 ^{c,§}	± 10.9				HDL = 0.88	
	HC-LHDL	118.9	± 11.7	116.4 [§]	± 10.8				D x HDL = 0.38	
	HP	122.7	± 13.9	119.5 [§]	± 13.6	121.1 [†]	± 0.7	T x D = 0.15		
	HC	118.3	± 11.5	115.6 ^{a,§}	± 10.9	117.1	± 0.7	T x HDL = 0.97		
	HHDL	120.7	± 13.2	117.8 [§]	± 13.0	119.0	± 0.6	T x D x HDL = 0.42		
	LHDL	120.9	± 12.9	117.9 [§]	± 12.2	119.2	± 0.8			
Time	120.8	± 13.1	117.8*	± 12.6						
HP = higher protein, HC = higher carbohydrate, HHDL = high HDL cholesterol, LHDL = low HDL cholesterol, T = time effect, D = diet effect, HDL = HDL risk factor effect, D x HDL = diet by HDL risk factor effect, T x D = time by diet effect, T x HDL = time by HDL risk factor effect, T x D x HDL = time by diet by HDL risk factor effect. Values are means ± standard deviations (except group means are means ± standard error) from 220 participants in the HP-HHDL group, 151 in the HP-LHDL group, 180 in the HC-HHDL group, 112 in the HC-LHDL group, 371 in the HP total group, 292 in the HC total group, 400 in the HHDL group, 263 in the LHDL group, and 663 participants total. * Significantly different than baseline, p < 0.05 (univariate). † Significant diet effect, p < 0.05 (univariate). ‡ Significant HDL effect, p < 0.05 (univariate). ^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than HHDL group, p < 0.05 (post hoc LSD). ^c Significantly different than HP-HHDL group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-LHDL group, p < 0.05 (post hoc LSD). ^e Significantly different than HC-HHDL group, p < 0.05 (post hoc LSD). ^f Significantly different than baseline, p < 0.05 (post hoc LSD).										

Table C.3: Resting Energy Expenditure After Ten Weeks of Exercise and Dietary Intervention Based on HDL Status

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Resting Energy Expenditure (kcal/day)	HP-HHDL	1669.2	± 284.7	1600.8 ^g	± 269.3				T = 0.001
	HP-LHDL	1737.7	± 282.1	1681.0 ^{c,g}	± 317.7				D = 0.001
	HC-HHDL	1585.9	± 242.9	1532.8 ^{c,g}	± 245.2				HDL = 0.003
	HC-LHDL	1624.4	± 255.3	1591.8 ^d	± 251.3				D x HDL = 0.53
	HP	1697.4	± 285.2	1633.8 ^e	± 292.5	1672.2 [†]	± 13.4		T x D = 0.26
	HC	1600.7	± 248.0	1555.5 ^{a,g}	± 248.8	1583.7	± 15.4		T x HDL = 0.36
	HHDL	1631.9	± 269.7	1570.4 ^{b,g}	± 260.7	1597.2	± 12.8		T x D x HDL = 0.80
	LHDL	1690.2	± 276.4	1643.6 ^e	± 294.5	1658.7 [‡]	± 15.9		
	Time	1655.2	± 273.7	1599.7 [*]	± 276.8				

HP = higher protein, HC = higher carbohydrate, HHDL = high HDL cholesterol, LHDL = low HDL cholesterol, T = time effect, D = diet effect, HDL = HDL risk factor effect, D x HDL = diet by HDL risk factor effect, T x D = time by diet effect, T x HDL = time by HDL risk factor effect, T x D x HDL = time by diet by HDL risk factor effect.

Values are means ± standard deviations (except group means are means ± standard error) from 210 participants in the HP-HHDL group, 147 in the HP-LHDL group, 170 in the HC-HHDL group, 106 in the HC-LHDL group, 357 in the HP total group, 276 in the HC total group, 380 in the HHDL group, 253 in the LHDL group, and 633 participants total.

* Significantly different than baseline, $p < 0.05$ (univariate). [†] Significant diet effect, $p < 0.05$ (univariate). [‡] Significant HDL effect, $p < 0.05$ (univariate).

^a Significantly different than HP group, $p < 0.05$ (post hoc LSD). ^b Significantly different than HHDL group, $p < 0.05$ (post hoc LSD).

^c Significantly different than HP-HHDL group, $p < 0.05$ (post hoc LSD). ^d Significantly different than HP-LHDL group, $p < 0.05$ (post hoc LSD).

^e Significantly different than HC-HHDL group, $p < 0.05$ (post hoc LSD). ^g Significantly different than baseline, $p < 0.05$ (post hoc LSD).

Table C.4: Resting Hemodynamic Measurements After Ten Weeks of Exercise and Dietary Intervention Based on HDL Status

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Resting Heart Rate (bpm)	HP-HHDL	71.0	± 10.2	68.5 [§]	± 9.6				T = 0.001
	HP-LHDL	73.5	± 10.4	69.6 [§]	± 9.8				D = 0.23
	HC-HHDL	70.6	± 10.4	68.2 [§]	± 10.7				HDL = 0.06
	HC-LHDL	72.1	± 10.7	68.2 [§]	± 8.6				D x HDL = 0.47
	HP	72.0	± 10.3	68.9 [§]	± 9.7	70.6	± 0.5		T x D = 0.88
	HC	71.2	± 10.5	68.2 [§]	± 9.9	69.8	± 0.5		T x HDL = 0.07
	HHDL	70.8	± 10.3	68.4 [§]	± 10.1	69.6	± 0.4		T x D x HDL = 0.92
	LHDL	72.9	± 10.5	69.0 [§]	± 9.3	70.9 [‡]	± 0.5		
	Time	71.6	± 10.4	68.6 [*]	± 9.8				
Resting Systolic Blood Pressure (mmHg)	HP-HHDL	126.0	± 15.4	123.4 [§]	± 13.8				T = 0.001
	HP-LHDL	125.0	± 13.7	122.4 [§]	± 13.6				D = 0.09
	HC-HHDL	124.2	± 13.9	122.8	± 14.6				HDL = 0.12
	HC-LHDL	124.4	± 15.3	118.5 ^{d,f,g}	± 13.0				D x HDL = 0.58
	HP	125.6	± 14.7	123.0 [§]	± 13.7	124.2	± 0.6		T x D = 0.39
	HC	124.3	± 14.4	121.2 ^{a,g}	± 14.1	122.5	± 0.7		T x HDL = 0.06
	HHDL	125.2	± 14.8	123.1 ^{b,g}	± 14.1	124.1	± 0.6		T x D x HDL = 0.06
	LHDL	124.7	± 14.3	120.7 [§]	± 13.5	122.6	± 0.8		
	Time	125.0	± 14.6	122.2 [*]	± 13.9				
Resting Diastolic Blood Pressure (mmHg)	HP-HHDL	81.1	± 9.0	80.1	± 8.8				T = 0.001
	HP-LHDL	81.5	± 9.6	78.8 [§]	± 8.8				D = 0.001
	HC-HHDL	79.7	± 9.3	77.8 ^{c,g}	± 8.8				HDL = 0.12
	HC-LHDL	78.8	± 9.5	75.7 ^{d,f,g}	± 9.6				D x HDL = 0.35
	HP	81.3	± 9.3	79.6 [§]	± 8.8	80.4 [†]	± 0.4		T x D = 0.43
	HC	79.3	± 9.4	77.0 ^{a,g}	± 9.2	78.0	± 0.5		T x HDL = 0.06
	HHDL	80.5	± 9.2	79.1 ^{b,g}	± 8.9	79.7	± 0.4		T x D x HDL = 0.77
	LHDL	80.3	± 9.6	77.5 [§]	± 9.3	78.7	± 0.5		
	Time	80.4	± 9.4	78.4 [*]	± 9.1				

HP = higher protein, HC = higher carbohydrate, HHDL = high HDL cholesterol, LHDL = low HDL cholesterol, T = time effect, D = diet effect, HDL = HDL risk factor effect, D x HDL = diet by HDL risk factor effect, T x D = time by diet effect, T x HDL = time by HDL risk factor effect, T x D x HDL = time by diet by HDL risk factor effect.

Values are means ± standard deviations (except group means are means ± standard error) from 220 participants in the HP-HHDL group, 151 in the HP-LHDL group, 180 in the HC-HHDL group, 112 in the HC-LHDL group, 371 in the HP total group, 292 in the HC total group, 400 in the HHDL group, 263 in the LHDL group, and 663 participants total.

* Significantly different than baseline, p < 0.05 (univariate). † Significant diet effect, p < 0.05 (univariate). ‡ Significant HDL effect, p < 0.05 (univariate).
^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than HHDL group, p < 0.05 (post hoc LSD).
^c Significantly different than HP-HHDL group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-LHDL group, p < 0.05 (post hoc LSD).
^e Significantly different than HC-HHDL group, p < 0.05 (post hoc LSD). ^f Significantly different than baseline, p < 0.05 (post hoc LSD).

Table C.5: Fasting Blood Lipid Values After Ten Weeks of Exercise and Dietary Intervention Based on HDL Status

Variable	Group	Baseline	10 Weeks	Group (SEM)	P-level
Total Cholesterol (mmol/L)	HP-HHDL	5.4 ± 0.9	5.1 ^g ± 1.0		T = 0.001
	HP-LHDL	4.9 ± 0.9	4.9 ^c ± 1.0		D = 0.68
	HC-HHDL	5.4 ± 1.0	5.1 ^g ± 0.9		HDL = 0.001
	HC-LHDL	4.9 ± 1.0	5.0 ± 1.0		D x HDL = 0.53
	HP	5.2 ± 1.0	5.0 ^g ± 1.0	5.1 ± 0.05	T x D = 0.37
	HC	5.2 ± 1.0	5.0 ^g ± 1.0	5.1 ± 0.05	T x HDL = 0.001
	HHDL	5.4 ± 1.0	5.1 ^{b,g} ± 0.9	5.2 ± 0.04	T x D x HDL = 0.96
	LHDL	4.9 ± 0.9	4.9 ± 1.0	4.9 [‡] ± 0.06	
	Time	5.2 ± 1.0	5.0* ± 1.0		
LDL (mmol/L)	HP-HHDL	3.2 ± 0.8	2.9 ^g ± 0.8		T = 0.001
	HP-LHDL	3.0 ± 0.7	3.0 ± 0.7		D = 0.42
	HC-HHDL	3.1 ± 0.9	3.0 ^g ± 0.8		HDL = 0.77
	HC-LHDL	3.1 ± 0.8	3.1 ± 0.8		D x HDL = 0.39
	HP	3.1 ± 0.8	3.0 ^g ± 0.8	3.0 ± 0.04	T x D = 0.67
	HC	3.1 ± 0.8	3.0 ^g ± 0.8	3.1 ± 0.04	T x HDL = 0.001
	HHDL	3.2 ± 0.8	2.9 ^g ± 0.8	3.0 ± 0.04	T x D x HDL = 0.66
	LHDL	3.1 ± 0.7	3.0 ± 0.8	3.1 ± 0.05	
	Time	3.1 ± 0.8	3.0* ± 0.8		
HDL (mmol/L)	HP-HHDL	1.6 ± 0.3	1.4 ^g ± 0.3		T = 0.001
	HP-LHDL	1.1 ± 0.1	1.1 ^c ± 0.2		D = 0.96
	HC-HHDL	1.6 ± 0.3	1.4 ^g ± 0.3		HDL = 0.001
	HC-LHDL	1.1 ± 0.1	1.1 ^f ± 0.2		D x HDL = 0.54
	HP	1.4 ± 0.3	1.3 ^g ± 0.3	1.3 ± 0.01	T x D = 0.60
	HC	1.4 ± 0.3	1.3 ^g ± 0.3	1.3 ± 0.01	T x HDL = 0.001
	HHDL	1.6 ± 0.3	1.4 ^{b,g} ± 0.3	1.5 ± 0.01	T x D x HDL = 0.88
	LHDL	1.1 ± 0.1	1.1 ^g ± 0.2	1.1 [‡] ± 0.01	
	Time	1.4 ± 0.3	1.3* ± 0.3		
Triglycerides (mmol/L)	HP-HHDL	1.5 ± 0.7	1.3 ^g ± 0.7		T = 0.001
	HP-LHDL	1.8 ± 0.9	1.6 ^{c,g} ± 0.9		D = 0.52
	HC-HHDL	1.4 ± 0.7	1.4 ± 0.7		HDL = 0.001
	HC-LHDL	1.7 ± 0.8	1.6 ^f ± 0.8		D x HDL = 0.72
	HP	1.6 ± 0.8	1.5 ^g ± 0.8	1.6 ± 0.04	T x D = 0.047
	HC	1.5 ± 0.7	1.5 ± 0.7	1.5 ± 0.04	T x HDL = 0.09
	HHDL	1.4 ± 0.7	1.4 ^{b,g} ± 0.7	1.4 ± 0.04	T x D x HDL = 0.94
	LHDL	1.8 ± 0.9	1.6 ^g ± 0.9	1.7 [‡] ± 0.05	
	Time	1.6 ± 0.8	1.5* ± 0.8		
TC/HDL ratio	HP-HHDL	3.5 ± 0.7	3.6 ^g ± 0.8		T = 0.18
	HP-LHDL	4.5 ± 0.8	4.4 ^{c,g} ± 0.9		D = 0.66
	HC-HHDL	3.5 ± 0.8	3.6 ^g ± 0.8		HDL = 0.001
	HC-LHDL	4.5 ± 1.0	4.5 ^f ± 1.1		D x HDL = 0.59
	HP	3.9 ± 0.9	3.9 ± 0.9	4.0 ± 0.04	T x D = 0.42
	HC	3.9 ± 1.0	4.0 ± 1.1	4.0 ± 0.05	T x HDL = 0.001
	HHDL	3.5 ± 0.7	3.6 ^{b,g} ± 0.8	3.6 ± 0.04	T x D x HDL = 0.42
	LHDL	4.5 ± 0.9	4.4 ± 1.0	4.5 [‡] ± 0.05	
	Time	3.9 ± 0.9	4.0 ± 1.0		

HP = higher protein, HC = higher carbohydrate, HHDL = high HDL cholesterol, LHDL = low HDL cholesterol, T = time effect, D = diet effect, HDL = HDL risk factor effect, D x HDL = diet by HDL risk factor effect, T x D = time by diet effect, T x HDL = time by HDL risk factor effect, T x D x HDL = time by diet by HDL risk factor effect.

Values are means ± standard deviations (except group means are means ± standard error) from 220 participants in the HP-HHDL group, 151 in the HP-LHDL group, 180 in the HC-HHDL group, 112 in the HC-LHDL group, 371 in the HP total group, 292 in the HC total group, 400 in the HHDL group, 263 in the LHDL group, and 663 participants total.

* Significantly different than baseline, $p < 0.05$ (univariate). † Significant diet effect, $p < 0.05$ (univariate). ‡ Significant HDL effect, $p < 0.05$ (univariate). ^a Significantly different than HP group, $p < 0.05$ (post hoc LSD). ^b Significantly different than HHDL group, $p < 0.05$ (post hoc LSD). ^c Significantly different than HP-HHDL group, $p < 0.05$ (post hoc LSD). ^d Significantly different than HP-LHDL group, $p < 0.05$ (post hoc LSD). ^e Significantly different than HC-HHDL group, $p < 0.05$ (post hoc LSD). ^f Significantly different than baseline, $p < 0.05$ (post hoc LSD).

Table C.6: Fasting Glucose and Insulin Values After Ten Weeks of Exercise and Dietary Intervention Based on HDL Status

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Glucose (mmol/L)	HP-HHDL	5.6	± 1.1	5.5 [§]	± 1.0				T = 0.032
	HP-LHDL	5.8	± 1.4	5.8 ^c	± 1.4				D = 0.23
	HC-HHDL	5.6	± 1.3	5.5	± 1.1				HDL = 0.18
	HC-LHDL	5.6	± 1.3	5.5	± 0.8				D x HDL = 0.14
	HP	5.7	± 1.2	5.6 [§]	± 1.2	5.7	± 0.06	T x D = 0.50	
	HC	5.6	± 1.3	5.5	± 1.0	5.6	± 0.07	T x HDL = 0.16	
	HHDL	5.6	± 1.2	5.5 ^{b,§}	± 1.1	5.6	± 0.06	T x D x HDL = 0.39	
	LHDL	5.7	± 1.4	5.7	± 1.2	5.7	± 0.07		
Time	5.7	± 1.3	5.6*	± 1.1					
Insulin (uIU/mL)	HP-HHDL	3.4	± 8.6	3.7	± 9.3				T = 0.79
	HP-LHDL	4.7	± 9.0	5.5	± 11.0				D = 0.001
	HC-HHDL	6.9	± 9.1	8.7 ^c	± 13.9				HDL = 0.25
	HC-LHDL	10.3	± 16.9	8.0	± 8.3				D x HDL = 0.93
	HP	4.0	± 8.8	4.5	± 10.1	4.3†	± 0.80	T x D = 0.52	
	HC	8.3	± 12.8	8.4 ^a	± 12.0	8.5	± 0.98	T x HDL = 0.13	
	HHDL	4.9	± 9.0	5.9	± 11.7	5.7	± 0.82	T x D x HDL = 0.06	
	LHDL	6.9	± 12.9	6.5	± 10.1	7.1	± 0.97		
Time	5.7	± 10.8	6.1	± 11.1					
Calculated HOMA	HP-HHDL	0.9	± 2.2	0.9	± 2.3				T = 0.93
	HP-LHDL	1.3	± 2.5	1.6	± 3.5				D = 0.002
	HC-HHDL	1.7	± 2.3	2.3 ^c	± 4.1				HDL = 0.12
	HC-LHDL	3.1	± 6.6	2.1 [§]	± 2.4				D x HDL = 0.97
	HP	1.0	± 2.3	1.2	± 2.9	1.2†	± 0.23	T x D = 0.37	
	HC	2.3	± 4.6	2.2 ^a	± 3.5	2.3	± 0.28	T x HDL = 0.14	
	HHDL	1.2	± 2.3	1.5	± 3.3	1.5	± 0.24	T x D x HDL = 0.030	
	LHDL	2.0	± 4.6	1.8	± 3.1	2.0	± 0.28		
Time	1.6	± 3.5	1.6	± 3.2					
HP = higher protein, HC = higher carbohydrate, HHDL = high HDL cholesterol, LHDL = low HDL cholesterol, T = time effect, D = diet effect, HDL = HDL risk factor effect, D x HDL = diet by HDL risk factor effect, T x D = time by diet effect, T x HDL = time by HDL risk factor effect, T x D x HDL = time by diet by HDL risk factor effect.									
Glucose values are means ± standard deviations (except group means are means ± standard error) from 220 participants in the HP-HHDL group, 151 in the HP-LHDL group, 180 in the HC-HHDL group, 112 in the HC-LHDL group, 371 in the HP total group, 292 in the HC total group, 400 in the HHDL group, 263 in the LHDL group, and 663 participants total. Insulin and HOMA values are means ± standard deviations (except group means are means ± standard error) from 83 participants in the HP-HHDL group, 66 in the HP-LHDL group, 62 in the HC-HHDL group, 41 in the HC-LHDL group, 149 in the HP total group, 103 in the HC total group, 145 in the HHDL group, 107 in the LHDL group, and 252 participants total.									
* Significantly different than baseline, p < 0.05 (univariate). † Significant diet effect, p < 0.05 (univariate). ‡ Significant HDL effect, p < 0.05 (univariate).									
^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than HHDL group, p < 0.05 (post hoc LSD).									
^c Significantly different than HP-HHDL group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-LHDL group, p < 0.05 (post hoc LSD).									
^e Significantly different than HC-HHDL group, p < 0.05 (post hoc LSD). [§] Significantly different than baseline, p < 0.05 (post hoc LSD).									

Table C.7: Fitness Parameters After Ten Weeks of Exercise and Dietary Intervention Based on HDL Status

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Bench Press Max Strength (1 RM / kg body weight)	HP-HHDL	30.3	± 7.4	32.4 ^g	± 7.2				T = 0.001
	HP-LHDL	31.8	± 7.8	33.7 ^g	± 7.6				D = 0.39
	HC-HHDL	28.5	± 7.2	30.5 ^{c,g}	± 7.2				HDL = 0.001
	HC-LHDL	32.1	± 8.3	35.0 ^{f,g}	± 8.8				D x HDL = 0.031
	HP	30.9	± 7.6	32.9 ^g	± 7.4	32.1	± 0.4		T x D = 0.25
	HC	29.9	± 7.8	32.2 ^c	± 8.1	31.5	± 0.5		T x HDL = 0.38
	HHDL	29.5	± 7.4	31.6 ^{b,g}	± 7.3	30.5	± 0.4		T x D x HDL = 0.20
	LHDL	31.9	± 8.0	34.2 ^g	± 8.2	33.1 [‡]	± 0.5		
	Time	30.5	± 7.7	32.6 [*]	± 7.7				
Bench Press Lift Volume (kg)	HP-HHDL	173.9	± 71.7	177.4	± 74.0				T = 0.94
	HP-LHDL	189.6	± 89.6	188.6	± 89.3				D = 0.48
	HC-HHDL	172.3	± 75.5	162.5	± 67.2				HDL = 0.001
	HC-LHDL	202.3	± 94.8	208.5 ^f	± 90.2				D x HDL = 0.031
	HP	180.3	± 79.7	182.0	± 80.7	182.4	± 3.7		T x D = 0.68
	HC	183.8	± 84.5	180.1	± 79.9	186.4	± 4.3		T x HDL = 0.44
	HHDL	173.2	± 73.3	170.9 ^b	± 71.4	171.5	± 3.6		T x D x HDL = 0.17
	LHDL	194.9	± 91.8	196.8	± 90.1	197.3 [‡]	± 4.4		
	Time	181.8	± 81.8	181.2	± 80.3				
Leg Press Max Strength (1 RM / kg body weight)	HP-HHDL	162.5	± 48.6	178.0 ^g	± 52.3				T = 0.001
	HP-LHDL	166.4	± 47.4	183.1 ^g	± 52.3				D = 0.31
	HC-HHDL	150.1	± 49.7	162.1 ^{c,g}	± 52.8				HDL = 0.001
	HC-LHDL	171.7	± 49.9	189.3 ^{f,g}	± 55.1				D x HDL = 0.016
	HP	164.1	± 48.1	180.1 ^g	± 52.3	172.5	± 2.7		T x D = 0.61
	HC	158.4	± 50.8	172.5 ^g	± 55.2	168.3	± 3.1		T x HDL = 0.17
	HHDL	157.1	± 49.4	171.0 ^{b,d}	± 53.0	163.2	± 2.6		T x D x HDL = 0.36
	LHDL	168.6	± 48.4	185.6 ^g	± 53.4	177.6 [‡]	± 3.2		
	Time	161.7	± 49.3	176.8 [*]	± 53.6				
Leg Press Lift Volume (kg)	HP-HHDL	1866.9	± 1045.3	1968.8	± 1122.7				T = 0.13
	HP-LHDL	1756.5	± 844.1	1881.4	± 1011.4				D = 0.016
	HC-HHDL	1582.7	± 842.0	1490.8 ^c	± 791.3				HDL = 0.14
	HC-LHDL	1803.6	± 886.5	1899.8 ^f	± 1009.8				D x HDL = 0.004
	HP	1821.8	± 968.3	1933.1 ^g	± 1077.9	1868.4 [†]	± 47.0		T x D = 0.14
	HC	1667.3	± 864.3	1647.4 ^a	± 901.7	1694.2	± 54.8		T x HDL = 0.16
	HHDL	1741.8	± 970.2	1758.4	± 1017.5	1727.3	± 45.3		T x D x HDL = 0.28
	LHDL	1776.0	± 860.4	1889.0	± 1008.6	1835.3	± 56.2		
	Time	1755.4	± 927.5	1810.3	± 1015.1				
Peak VO₂ (mL/kg/min)	HP-HHDL	20.0	± 4.5	21.7 ^g	± 4.4				T = 0.002
	HP-LHDL	20.0	± 4.6	22.3 ^g	± 4.6				D = 0.015
	HC-HHDL	20.4	± 4.4	22.8 ^{c,g}	± 4.8				HDL = 0.30
	HC-LHDL	20.6	± 3.9	23.5 ^{d,g}	± 4.6				D x HDL = 0.80
	HP	20.0	± 4.5	21.9 ^g	± 4.5	21.0 [†]	± 0.22		T x D = 0.012
	HC	20.5	± 4.2	23.1 ^{a,g}	± 4.7	21.8	± 0.25		T x HDL = 0.024
	HHDL	20.2	± 4.4	22.2 ^g	± 4.6	21.2	± 0.21		T x D x HDL = 0.98
	LHDL	20.2	± 4.3	22.8 ^g	± 4.6	21.6	± 0.26		
	Time	20.2	± 4.4	22.4 [*]	± 4.6				

HP = higher protein, HC = higher carbohydrate, HHDL = high HDL cholesterol, LHDL = low HDL cholesterol, T = time effect, D = diet effect, HDL = HDL risk factor effect, D x HDL = diet by HDL risk factor effect, T x D = time by diet effect, T x HDL = time by HDL risk factor effect, T x D x HDL = time by diet by HDL risk factor effect.

Strength values are means ± standard deviations (except group means are means ± standard error) from 201 participants in the HP-HHDL group, 139 in the HP-LHDL group, 158 in the HC-HHDL group, 98 in the HC-LHDL group, 340 in the HP total group, 256 in the HC total group, 359 in the HHDL group, 237 in the LHDL group, and 596 participants total. Peak VO₂ values are means ± standard deviations (except group means are means ± standard error) from 220 participants in the HP-HHDL group, 151 in the HP-LHDL group, 180 in the HC-HHDL group, 112 in the HC-LHDL group, 371 in the HP total group, 292 in the HC total group, 400 in the HHDL group, 263 in the LHDL group, and 663 participants total.

* Significantly different than baseline, p < 0.05 (univariate). † Significant diet effect, p < 0.05 (univariate). ‡ Significant HDL effect, p < 0.05 (univariate).
^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than HHDL group, p < 0.05 (post hoc LSD).
^c Significantly different than HP-HHDL group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-LHDL group, p < 0.05 (post hoc LSD).
^f Significantly different than HC-HHDL group, p < 0.05 (post hoc LSD). ^g Significantly different than baseline, p < 0.05 (post hoc LSD).

Table C.8: Changes in SF36 - Quality of Life Inventory Values After Ten Weeks of Exercise and Dietary Intervention Based on HDL Status

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Physical Functioning	HP-HHDL	76.9	± 27.6	81.8 ^a	± 24.9				T = 0.001
	HP-LHDL	74.0	± 32.7	80.5 ^a	± 24.7				D = 0.61
	HC-HHDL	77.5	± 17.4	81.4	± 18.0				HDL = 0.75
	HC-LHDL	74.6	± 26.1	85.0 ^a	± 23.2				D x HDL = 0.64
	HP	75.7	± 29.7	81.3 ^a	± 24.7	78.3	± 1.79		T x D = 0.61
	HC	76.4	± 21.2	82.8 ^a	± 20.2	79.6	± 1.90		T x HDL = 0.14
	HHDL	77.2	± 23.2	81.6 ^a	± 21.8	79.4	± 1.66		T x D x HDL = 0.38
	LHDL	74.3	± 29.7	82.6 ^a	± 24.0	78.5	± 2.01		
	Time	76.0	± 26.0	82.0*	± 22.7				
Role Physical	HP-HHDL	147.5	± 136.0	158.5	± 151.9				T = 0.42
	HP-LHDL	134.6	± 140.4	129.4	± 141.7				D = 0.001
	HC-HHDL	265.5	± 137.2	268.8 ^c	± 138.6				HDL = 0.06
	HC-LHDL	222.7	± 147.4	227.0 ^d	± 157.3				D x HDL = 0.53
	HP	142.2	± 137.5	146.4	± 148.0	142.5†	± 11.45		T x D = 0.92
	HC	248.6	± 142.3	252.3 ^a	± 147.1	246.0	± 12.16		T x HDL = 0.36
	HHDL	204.5	± 148.5	211.7 ^b	± 155.4	210.1	± 10.61		T x D x HDL = 0.30
	LHDL	175.3	± 149.7	174.4	± 156.3	178.4	± 12.90		
	Time	192.6	± 149.4	196.6	± 156.5				
Bodily Pain	HP-HHDL	58.8	± 19.0	62.0	± 20.5				T = 0.004
	HP-LHDL	59.4	± 19.1	64.8 ^a	± 21.1				D = 0.35
	HC-HHDL	60.2	± 21.4	59.8	± 21.2				HDL = 0.06
	HC-LHDL	64.7	± 21.4	68.7 ^f	± 21.5				D x HDL = 0.26
	HP	59.1	± 19.0	63.1 ^a	± 20.7	61.2	± 1.54		T x D = 0.23
	HC	62.0	± 21.4	63.3	± 21.6	63.4	± 1.64		T x HDL = 0.11
	HHDL	59.5	± 20.1	60.9 ^b	± 20.8	60.2	± 1.43		T x D x HDL = 0.58
	LHDL	61.9	± 20.3	66.6 ^a	± 21.2	64.4	± 1.74		
	Time	60.5	± 20.2	63.2*	± 21.1				
General Health	HP-HHDL	51.0	± 28.5	55.9 ^a	± 29.2				T = 0.001
	HP-LHDL	49.5	± 28.9	53.2 ^a	± 27.9				D = 0.001
	HC-HHDL	68.1	± 20.3	71.3 ^{c,g}	± 20.0				HDL = 0.11
	HC-LHDL	60.5	± 24.6	63.8 ^{d,g}	± 24.6				D x HDL = 0.37
	HP	50.4	± 28.6	54.8 ^a	± 28.6	52.4†	± 2.06		T x D = 0.47
	HC	65.1	± 22.3	68.4 ^{a,g}	± 22.1	65.9	± 2.19		T x HDL = 0.66
	HHDL	59.2	± 26.3	63.4 ^a	± 26.3	61.6	± 1.91		T x D x HDL = 0.64
	LHDL	54.6	± 27.5	58.1 ^a	± 26.8	56.8	± 2.32		
	Time	57.3	± 26.8	61.2*	± 26.6				
Vital	HP-HHDL	37.4	± 22.9	45.7 ^a	± 28.0				T = 0.001
	HP-LHDL	46.6	± 66.2	54.0 ^a	± 65.9				D = 0.12
	HC-HHDL	51.0	± 17.5	57.0 ^{c,g}	± 15.8				HDL = 0.48
	HC-LHDL	49.4	± 20.0	53.3 ^a	± 22.1				D x HDL = 0.18
	HP	41.3	± 46.1	49.1 ^a	± 47.5	45.9	± 2.92		T x D = 0.11
	HC	50.4	± 18.5	55.5 ^a	± 18.6	52.7	± 3.10		T x HDL = 0.40
	HHDL	44.0	± 21.5	51.2 ^a	± 23.6	47.8	± 2.71		T x D x HDL = 0.75
	LHDL	47.9	± 50.2	53.7 ^a	± 50.5	50.8	± 3.29		
	Time	45.6	± 36.0	52.2*	± 36.8				

Table C.8: Continued

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Social Functioning	HP-HHDL	47.2	± 26.7	53.3 [§]	± 27.5				T = 0.024
	HP-LHDL	44.9	± 24.5	47.0	± 25.4				D = 0.001
	HC-HHDL	62.4	± 24.0	64.2 ^c	± 25.0				HDL = 0.11
	HC-LHDL	54.4	± 25.7	53.8 ^f	± 22.6				D x HDL = 0.39
	HP	46.2	± 25.7	50.7 [§]	± 26.7	48.1 [†]	± 1.96	T x D = 0.09	
	HC	59.3	± 24.9	60.1 ^a	± 24.5	58.7	± 2.08	T x HDL = 0.11	
	HHDL	54.5	± 26.4	58.6 ^{b,§}	± 26.8	56.8	± 1.82	T x D x HDL = 0.69	
	LHDL	49.3	± 25.4	50.1	± 24.3	50.0	± 2.21		
	Time	52.4	± 26.1	55.2 [*]	± 26.1				
Role Emotional	HP-HHDL	224.1	± 123.1	249.1	± 140.8				T = 0.45
	HP-LHDL	210.1	± 136.9	223.8	± 140.1				D = 0.001
	HC-HHDL	310.7	± 220.7	292.6 ^c	± 132.8				HDL = 0.22
	HC-LHDL	280.0	± 130.5	284.4 ^d	± 136.7				D x HDL = 1.00
	HP	218.3	± 128.7	238.6	± 140.6	226.7 [†]	± 10.95	T x D = 0.12	
	HC	298.6	± 190.3	289.4 ^a	± 133.9	291.9	± 11.63	T x HDL = 0.73	
	HHDL	265.9	± 181.8	270.1	± 138.3	269.1	± 10.15	T x D x HDL = 0.31	
	LHDL	242.3	± 137.9	251.8	± 141.2	249.6	± 12.34		
	Time	256.3	± 165.6	262.7	± 139.6				
Mental Health	HP-HHDL	55.9	± 16.6	65.3 [§]	± 15.0				T = 0.001
	HP-LHDL	57.4	± 14.0	64.9 [§]	± 15.9				D = 0.035
	HC-HHDL	60.5	± 14.9	66.7 [§]	± 12.0				HDL = 0.65
	HC-LHDL	59.8	± 13.7	69.0 [§]	± 12.2				D x HDL = 0.93
	HP	56.5	± 15.5	65.1 [§]	± 15.3	60.9 [†]	± 1.02	T x D = 0.67	
	HC	60.3	± 14.4	67.6 [§]	± 12.1	64.0	± 1.08	T x HDL = 0.75	
	HHDL	58.2	± 15.9	66.0 [§]	± 13.6	62.1	± 0.95	T x D x HDL = 0.17	
	LHDL	58.5	± 13.8	66.8 [§]	± 14.4	62.8	± 1.15		
	Time	58.3	± 15.1	66.3 [*]	± 13.9				
HP = higher protein, HC = higher carbohydrate, HHDL = high HDL cholesterol, LHDL = low HDL cholesterol, T = time effect, D = diet effect, HDL = HDL risk factor effect, D x HDL = diet by HDL risk factor effect, T x D = time by diet effect, T x HDL = time by HDL risk factor effect, T x D x HDL = time by diet by HDL risk factor effect. Values are means ± standard deviations (except group means are means ± standard error) from 89 participants in the HP-HHDL group, 63 in the HP-LHDL group, 83 in the HC-HHDL group, 54 in the HC-LHDL group, 152 in the HP total group, 137 in the HC total group, 172 in the HHDL group, 117 in the LHDL group, and 289 participants total. * Significantly different than baseline, p < 0.05 (univariate). † Significant diet effect, p < 0.05 (univariate). ‡ Significant HDL effect, p < 0.05 (univariate). ^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than HHDL group, p < 0.05 (post hoc LSD). ^c Significantly different than HP-HHDL group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-LHDL group, p < 0.05 (post hoc LSD). ^e Significantly different than HC-HHDL group, p < 0.05 (post hoc LSD). ^f Significantly different than baseline, p < 0.05 (post hoc LSD).									

Table C.9: Changes in Body Image Evaluation After Ten Weeks of Exercise and Dietary Intervention Based on HDL Status

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Appearance Evaluation	HP-HHDL	2.4	± 0.6	2.7 ^g	± 0.7				T = 0.001
	HP-LHDL	2.5	± 0.6	2.8 ^g	± 0.6				D = 0.58
	HC-HHDL	2.5	± 0.6	2.8 ^g	± 0.7				HDL = 1.00
	HC-LHDL	2.4	± 0.7	2.8 ^g	± 0.8				D x HDL = 0.30
	HP	2.4	± 0.6	2.7 ^g	± 0.7	2.6	± 0.04		T x D = 0.46
	HC	2.4	± 0.6	2.8 ^g	± 0.7	2.6	± 0.04		T x HDL = 0.78
	HHDL	2.4	± 0.6	2.8 ^g	± 0.7	2.6	± 0.04		T x D x HDL = 0.42
	LHDL	2.4	± 0.7	2.8 ^g	± 0.7	2.6	± 0.04		
	Time	2.4	± 0.6	2.8*	± 0.7				
Appearance Orientation	HP-HHDL	4.1	± 0.9	4.1	± 0.9				T = 0.20
	HP-LHDL	4.2	± 0.8	4.1	± 0.9				D = 0.010
	HC-HHDL	4.0	± 0.8	3.9	± 0.9				HDL = 0.83
	HC-LHDL	3.9	± 0.8	3.8 ^d	± 0.8				D x HDL = 0.48
	HP	4.1	± 0.9	4.1	± 0.9	4.1 [†]	± 0.05		T x D = 0.35
	HC	3.9	± 0.8	3.9 ^a	± 0.9	3.9	± 0.06		T x HDL = 0.59
	HHDL	4.0	± 0.9	4.0	± 0.9	4.0	± 0.05		T x D x HDL = 0.52
	LHDL	4.1	± 0.8	4.0	± 0.9	4.0	± 0.06		
	Time	4.1	± 0.8	4.0	± 0.9				
Body Area Satisfaction	HP-HHDL	2.1	± 0.7	2.3 ^g	± 0.8				T = 0.001
	HP-LHDL	2.0	± 0.8	2.2 ^g	± 0.9				D = 0.001
	HC-HHDL	2.2	± 0.7	2.6 ^{c,g}	± 0.8				HDL = 0.55
	HC-LHDL	2.2	± 0.7	2.5 ^{d,g}	± 0.9				D x HDL = 0.62
	HP	2.0	± 0.8	2.3 ^g	± 0.8	2.1 [†]	± 0.05		T x D = 0.20
	HC	2.2	± 0.7	2.5 ^{a,g}	± 0.8	2.4	± 0.06		T x HDL = 0.75
	HHDL	2.1	± 0.7	2.4 ^g	± 0.8	2.3	± 0.05		T x D x HDL = 0.94
	LHDL	2.1	± 0.8	2.4 ^g	± 0.9	2.2	± 0.06		
	Time	2.1	± 0.7	2.4*	± 0.8				
Overweight Preoccupation	HP-HHDL	2.9	± 0.7	3.4 ^g	± 0.8				T = 0.001
	HP-LHDL	2.8	± 0.7	3.4 ^g	± 0.6				D = 0.30
	HC-HHDL	2.9	± 0.7	3.3 ^g	± 0.7				HDL = 0.91
	HC-LHDL	2.9	± 0.8	3.3 ^g	± 0.7				D x HDL = 0.76
	HP	2.9	± 0.7	3.4 ^g	± 0.7	3.1	± 0.04		T x D = 0.09
	HC	2.9	± 0.7	3.3 ^g	± 0.7	3.1	± 0.05		T x HDL = 0.29
	HHDL	2.9	± 0.7	3.3 ^g	± 0.7	3.1	± 0.04		T x D x HDL = 0.18
	LHDL	2.8	± 0.7	3.4 ^g	± 0.7	3.1	± 0.05		
	Time	2.9	± 0.7	3.3*	± 0.7				
Self-Classified Weight	HP-HHDL	4.2	± 0.8	4.2	± 0.6				T = 0.007
	HP-LHDL	4.3	± 0.6	4.1	± 0.7				D = 0.11
	HC-HHDL	4.1	± 0.7	4.0 ^c	± 0.7				HDL = 0.53
	HC-LHDL	4.2	± 0.6	4.1	± 0.7				D x HDL = 0.33
	HP	4.2	± 0.7	4.2	± 0.7	4.2	± 0.04		T x D = 0.57
	HC	4.2	± 0.7	4.0 ^g	± 0.7	4.1	± 0.04		T x HDL = 0.21
	HHDL	4.2	± 0.8	4.1	± 0.7	4.1	± 0.04		T x D x HDL = 0.92
	LHDL	4.3	± 0.6	4.1 ^g	± 0.7	4.2	± 0.04		
	Time	4.2	± 0.7	4.1*	± 0.7				

Table C.9: Continued

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Rosenberg Self Esteem	HP-HHDL	25.4	± 3.7	26.1 ^a	± 3.6				T = 0.008
	HP-LHDL	26.1	± 4.3	26.6	± 4.2				D = 0.55
	HC-HHDL	25.6	± 3.7	25.7	± 3.5				HDL = 0.16
	HC-LHDL	25.8	± 4.3	26.3	± 4.6				D x HDL = 0.77
	HP	25.7	± 3.9	26.3 ^a	± 3.8	26.1	± 0.23	T x D = 0.29	
	HC	25.7	± 3.9	25.9	± 4.0	25.9	± 0.26	T x HDL = 0.81	
	HHDL	25.5	± 3.7	25.9 ^a	± 3.6	25.7	± 0.22	T x D x HDL = 0.42	
	LHDL	26.0	± 4.3	26.5	± 4.3	26.2	± 0.27		
Time	25.7	± 3.9	26.2 [*]	± 3.9					
Social Physique Anxiety	HP-HHDL	31.5	± 6.0	31.5	± 6.5				T = 0.58
	HP-LHDL	30.8	± 6.7	31.2	± 5.8				D = 0.080
	HC-HHDL	30.6	± 5.8	30.5	± 5.0				HDL = 0.19
	HC-LHDL	32.0	± 6.2	32.5 ^f	± 7.4				D x HDL = 0.025
	HP	31.3	± 6.3	31.4	± 6.2	31.3	± 0.32	T x D = 0.91	
	HC	31.1	± 6.0	31.3	± 6.1	31.4	± 0.38	T x HDL = 0.40	
	HHDL	31.1	± 5.9	31.0	± 5.9	31.0	± 0.31	T x D x HDL = 0.90	
	LHDL	31.3	± 6.5	31.8	± 6.5	31.6	± 0.38		
Time	31.2	± 6.1	31.3	± 6.2					
HP = higher protein, HC = higher carbohydrate, HHDL = high HDL cholesterol, LHDL = low HDL cholesterol, T = time effect, D = diet effect, HDL = HDL risk factor effect, D x HDL = diet by HDL risk factor effect, T x D = time by diet effect, T x HDL = time by HDL risk factor effect, T x D x HDL = time by diet by HDL risk factor effect. Values are means ± standard deviations (except group means are means ± standard error) from 150 participants in the HP-HHDL group, 106 in the HP-LHDL group, 120 in the HC-HHDL group, 75 in the HC-LHDL group, 256 in the HP total group, 195 in the HC total group, 270 in the HHDL group, 181 in the LHDL group, and 451 participants total. [*] Significantly different than baseline, $p < 0.05$ (univariate). [†] Significant diet effect, $p < 0.05$ (univariate). [‡] Significant HDL effect, $p < 0.05$ (univariate). ^a Significantly different than HP group, $p < 0.05$ (post hoc LSD). ^b Significantly different than HHDL group, $p < 0.05$ (post hoc LSD). ^c Significantly different than HP-HHDL group, $p < 0.05$ (post hoc LSD). ^d Significantly different than HP-LHDL group, $p < 0.05$ (post hoc LSD). ^e Significantly different than HC-HHDL group, $p < 0.05$ (post hoc LSD). ^f Significantly different than baseline, $p < 0.05$ (post hoc LSD).									

APPENDIX D

BLOOD PRESSURE RISK FACTOR TABLES

Table D.1: Body Composition Before and After Ten Weeks of Exercise and Dietary Intervention Based on Blood Pressure Status and Measured via DEXA Scan

Variable	Group	Baseline			10 Weeks			Group (SEM)			P-level
Scanned Mass (kg)	HP-LBP	87.5	±	18.0	83.7 ^g	±	17.7				T = 0.001
	HP-HBP	91.9	±	18.0	87.7 ^{c-g}	±	16.9				D = 0.001
	HC-LBP	81.6	±	13.7	78.8 ^{c-g}	±	13.1				BP = 0.014
	HC-HBP	84.1	±	15.5	80.8 ^{d-g}	±	14.8				D x BP = 0.47
	HP	89.2	±	18.1	85.3 ^g	±	17.5	87.7 [†]	±	0.85	T x D = 0.002
	HC	82.5	±	14.3	79.5 ^{a-g}	±	13.7	81.4	±	1.00	T x BP = 0.17
	LBP	84.8	±	16.4	81.4 ^{b-g}	±	15.9	82.9	±	0.79	T x D x BP = 0.94
	HBP	88.8	±	17.4	85.0 ^g	±	16.4	86.1 [‡]	±	1.05	
Time	86.2	±	16.9	82.7 [*]	±	16.2					
Fat Mass (kg)	HP-LBP	40.2	±	11.2	37.1 ^g	±	11.2				T = 0.001
	HP-HBP	42.5	±	11.8	39.3 ^{c-g}	±	11.1				D = 0.001
	HC-LBP	36.4	±	8.7	34.2 ^{c-g}	±	8.3				BP = 0.018
	HC-HBP	38.4	±	9.8	35.6 ^{d-g}	±	9.5				D x BP = 0.75
	HP	41.1	±	11.4	38.0 ^g	±	11.2	39.8 [†]	±	0.54	T x D = 0.007
	HC	37.1	±	9.1	34.7 ^{a-g}	±	8.7	36.2	±	0.64	T x BP = 0.13
	LBP	38.4	±	10.2	35.8 ^{b-g}	±	10.0	37.0	±	0.50	T x D x BP = 0.32
	HBP	40.9	±	11.2	37.9 ^g	±	10.6	39.0 [‡]	±	0.67	
Time	39.3	±	10.7	36.5 [*]	±	10.3					
Lean Mass (kg)	HP-LBP	45.5	±	7.8	44.8 ^g	±	7.6				T = 0.001
	HP-HBP	47.5	±	7.5	46.6 ^{c-g}	±	6.9				D = 0.001
	HC-LBP	43.4	±	6.3	42.8 ^{c-g}	±	6.0				BP = 0.029
	HC-HBP	44.0	±	6.7	43.5 ^{d-g}	±	6.5				D x BP = 0.26
	HP	46.3	±	7.7	45.5 ^g	±	7.4	46.1 [†]	±	0.37	T x D = 0.11
	HC	43.6	±	6.4	43.0 ^{a-g}	±	6.2	43.4	±	0.43	T x BP = 0.78
	LBP	44.5	±	7.2	43.9 ^{b-g}	±	7.0	44.1	±	0.34	T x D x BP = 0.22
	HBP	46.1	±	7.4	45.3 ^g	±	6.9	45.4 [‡]	±	0.46	
Time	45.1	±	7.3	44.4 [*]	±	7.0					
Body Fat (%)	HP-LBP	45.5	±	4.2	43.8 ^g	±	4.7				T = 0.001
	HP-HBP	45.6	±	4.7	44.1 ^g	±	4.8				D = 0.11
	HC-LBP	44.4	±	4.3	43.2 ^g	±	4.3				BP = 0.15
	HC-HBP	45.4	±	3.8	43.7 ^g	±	4.2				D x BP = 0.42
	HP	45.5	±	4.4	43.9 ^g	±	4.7	44.7	±	0.23	T x D = 0.41
	HC	44.7	±	4.2	43.4 ^g	±	4.3	44.2	±	0.27	T x BP = 0.52
	LBP	45.0	±	4.3	43.5 ^g	±	4.5	44.2	±	0.21	T x D x BP = 0.045
	HBP	45.5	±	4.4	44.0 ^g	±	4.6	44.7	±	0.28	
Time	45.2	±	4.3	43.7 [*]	±	4.5					
HP = higher protein, HC = higher carbohydrate, LBP = low blood pressure, HBP = high blood pressure, T = time effect, D = diet effect, BP = blood pressure risk factor effect, D x BP = diet by blood pressure risk factor effect, T x D = time by diet effect, T x BP = time by blood pressure risk factor effect, T x D x BP = time by diet by blood pressure risk factor effect. Values are means ± standard deviations (except group means are means ± standard error) from 223 participants in the HP-LBP group, 148 in the HP-HBP group, 196 in the HC-LBP group, 96 in the HC-HBP group, 371 in the HP total group, 292 in the HC total group, 419 in the LBP group, 244 in the HBP group, and 663 participants total. * Significantly different than baseline, p < 0.05 (univariate). † Significant diet effect, p < 0.05 (univariate). ‡ Significant blood pressure effect, p < 0.05 (univariate). ^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than LBP group, p < 0.05 (post hoc LSD). ^c Significantly different than HP-LBP group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-HBP group, p < 0.05 (post hoc LSD). ^e Significantly different than HC-LBP group, p < 0.05 (post hoc LSD). ^g Significantly different than baseline, p < 0.05 (post hoc LSD).											

Table D.2: Body Composition Before and After Ten Weeks of Exercise and Dietary Intervention Based on Blood Pressure Status and Measured via Anthropometric Measurements

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Weight (kg)	HP-LBP	94.0	± 19.0	89.8 ^g	± 18.6				T = 0.001
	HP-HBP	98.5	± 18.9	94.0 ^{c-g}	± 17.7				D = 0.001
	HC-LBP	87.9	± 14.1	84.9 ^{c-g}	± 13.5				BP = 0.016
	HC-HBP	90.5	± 16.3	86.8 ^{d-g}	± 15.6				D x BP = 0.46
	HP	95.8	± 19.1	91.5 ^g	± 18.3	94.1 [†]	± 0.9	T x D = 0.001	
	HC	88.8	± 14.9	85.5 ^{a-g}	± 14.2	87.5	± 1.0	T x BP = 0.07	
	LBP	91.1	± 17.1	87.5 ^{b-g}	± 16.6	89.1	± 0.8	T x D x BP = 0.63	
	HBP	95.4	± 18.3	91.2 ^g	± 17.3	92.5 [‡]	± 1.1		
Time	92.7	± 17.7	88.9 [*]	± 16.9					
Body Mass Index (kg/m ²)	HP-LBP	35.3	± 6.6	33.7 ^g	± 6.5				T = 0.001
	HP-HBP	36.9	± 6.4	35.3 ^{c-g}	± 6.1				D = 0.001
	HC-LBP	33.0	± 5.0	31.9 ^{c-g}	± 4.7				BP = 0.013
	HC-HBP	33.9	± 5.7	32.5 ^{d-g}	± 5.4				D x BP = 0.38
	HP	35.9	± 6.6	34.3 ^g	± 6.4	35.3 [†]	± 0.3	T x D = 0.001	
	HC	33.3	± 5.2	32.1 ^{a-g}	± 5.0	32.8	± 0.4	T x BP = 0.085	
	LBP	34.2	± 6.0	32.9 ^{b-g}	± 5.8	33.5	± 0.3	T x D x BP = 0.59	
	HBP	35.7	± 6.3	34.2 ^g	± 6.0	34.7 [‡]	± 0.4		
Time	34.8	± 6.2	33.3 [*]	± 5.9					
Waist Circumference (cm)	HP-LBP	99.5	± 13.1	95.6 ^g	± 13.0				T = 0.001
	HP-HBP	104.1	± 13.5	99.9 ^{c-g}	± 13.0				D = 0.001
	HC-LBP	94.7	± 10.9	91.7 ^{c-g}	± 10.1				BP = 0.001
	HC-HBP	97.9	± 12.9	94.5 ^{d-g}	± 13.1				D x BP = 0.45
	HP	101.3	± 13.5	97.3 ^g	± 13.2	99.8 [†]	± 0.6	T x D = 0.10	
	HC	95.8	± 11.7	92.6 ^{a-g}	± 11.2	94.7	± 0.8	T x BP = 0.48	
	LBP	97.3	± 12.4	93.7 ^{b-g}	± 11.9	95.4	± 0.6	T x D x BP = 0.89	
	HBP	101.7	± 13.6	97.8 ^g	± 13.2	99.1 [‡]	± 0.8		
Time	98.9	± 13.0	95.2 [*]	± 12.6					
Hip Circumference (cm)	HP-LBP	121.6	± 13.4	118.3 ^g	± 13.5				T = 0.001
	HP-HBP	124.4	± 14.5	121.4 ^{c-g}	± 13.6				D = 0.001
	HC-LBP	117.3	± 11.0	115.0 ^{c-g}	± 10.5				BP = 0.005
	HC-HBP	120.5	± 12.3	117.0 ^{d-g}	± 11.5				D x BP = 0.89
	HP	122.7	± 13.9	119.5 ^g	± 13.6	121.4 [†]	± 0.7	T x D = 0.51	
	HC	118.3	± 11.5	115.6 ^{a-g}	± 10.9	117.5	± 0.8	T x BP = 0.29	
	LBP	119.6	± 12.5	116.7 ^{b-g}	± 12.3	118.0	± 0.6	T x D x BP = 0.07	
	HBP	122.9	± 13.8	119.7 ^g	± 13.0	120.9 [‡]	± 0.8		
Time	120.8	± 13.1	117.8 [*]	± 12.6					
HP = higher protein, HC = higher carbohydrate, LBP = low blood pressure, HBP = high blood pressure, T = time effect, D = diet effect, BP = blood pressure risk factor effect, D x BP = diet by blood pressure risk factor effect, T x D = time by diet effect, T x BP = time by blood pressure risk factor effect. Values are means ± standard deviations (except group means are means ± standard error) from 223 participants in the HP-LBP group, 148 in the HP-HBP group, 196 in the HC-LBP group, 96 in the HC-HBP group, 371 in the HP total group, 292 in the HC total group, 419 in the LBP group, 244 in the HBP group, and 663 participants total. * Significantly different than baseline, p < 0.05 (univariate). † Significant diet effect, p < 0.05 (univariate). ‡ Significant blood pressure effect, p < 0.05 (univariate). ^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than LBP group, p < 0.05 (post hoc LSD). ^c Significantly different than HP-LBP group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-HBP group, p < 0.05 (post hoc LSD). ^e Significantly different than HC-LBP group, p < 0.05 (post hoc LSD). ^g Significantly different than baseline, p < 0.05 (post hoc LSD).									

Table D.3: Resting Energy Expenditure After Ten Weeks of Exercise and Dietary Intervention Based on Blood Pressure Status

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Resting Energy Expenditure (kcal/day)	HP-LBP	1662.6	± 272.3	1609.8 ^g	± 274.9				T = 0.001
	HP-HBP	1748.2	± 296.9	1668.9 ^{c,g}	± 314.1				D = 0.001
	HC-LBP	1588.2	± 245.1	1539.4 ^{c,g}	± 245.8				BP = 0.005
	HC-HBP	1627.3	± 253.4	1589.8 ^d	± 253.1				D x BP = 0.51
	HP	1697.4	± 285.2	1633.8 ^e	± 292.5	1672.4 [†]	± 13.4		T x D = 0.20
	HC	1600.7	± 248.0	1555.5 ^{a,g}	± 248.8	1586.2	± 16.1		T x BP = 0.67
	LBP	1627.6	± 262.2	1576.7 ^{b,g}	± 263.6	1600.0	± 12.5		T x D x BP = 0.29
	HBP	1702.6	± 286.8	1639.0 ^g	± 294.5	1658.6 [‡]	± 16.8		
	Time	1655.2	± 273.7	1599.7 [*]	± 276.8				

HP = higher protein, HC = higher carbohydrate, LBP = low blood pressure, HBP = high blood pressure, T = time effect, D = diet effect, BP = blood pressure risk factor effect, D x BP = diet by blood pressure risk factor effect, T x D = time by diet effect, T x BP = time by blood pressure risk factor effect, T x D x BP = time by diet by blood pressure risk factor effect.

Values are means ± standard deviations (except group means are means ± standard error) from 212 participants in the HP-LBP group, 145 in the HP-HBP group, 188 in the HC-LBP group, 88 in the HC-HBP group, 357 in the HP total group, 276 in the HC total group, 400 in the LBP group, 233 in the HBP group, and 633 participants total.

* Significantly different than baseline, $p < 0.05$ (univariate). [†] Significant diet effect, $p < 0.05$ (univariate). [‡] Significant blood pressure effect, $p < 0.05$ (univariate). ^a Significantly different than HP group, $p < 0.05$ (post hoc LSD). ^b Significantly different than LBP group, $p < 0.05$ (post hoc LSD). ^c Significantly different than HP-LBP group, $p < 0.05$ (post hoc LSD). ^d Significantly different than HP-HBP group, $p < 0.05$ (post hoc LSD). ^e Significantly different than HC-LBP group, $p < 0.05$ (post hoc LSD). ^f Significantly different than baseline, $p < 0.05$ (post hoc LSD).

Table D.4: Resting Hemodynamic Measurements After Ten Weeks of Exercise and Dietary Intervention Based on Blood Pressure Status

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Resting Heart Rate (bpm)	HP-LBP	72.0	± 9.6	69.0 [§]	± 9.5				T = 0.001
	HP-HBP	72.1	± 11.3	68.7 [§]	± 10.0				D = 0.63
	HC-LBP	70.3	± 10.1	67.5 [§]	± 10.1				BP = 0.11
	HC-HBP	72.9	± 11.2	69.7 [§]	± 9.5				D x BP = 0.08
	HP	72.0	± 10.3	68.9 [§]	± 9.7	70.4	± 0.5	T x D = 0.87	
	HC	71.2	± 10.5	68.2 [§]	± 9.9	70.1	± 0.5	T x BP = 0.61	
	LBP	71.2	± 9.9	68.3 [§]	± 9.8	69.7	± 0.4	T x D x BP = 0.93	
	HBP	72.4	± 11.2	69.1 [§]	± 9.8	70.9	± 0.6		
Time	71.6	± 10.4	68.6*	± 9.8					
Resting Systolic Blood Pressure (mmHg)	HP-LBP	117.5	± 8.8	119.0	± 11.3				T = 0.001
	HP-HBP	137.7	± 13.5	128.9 ^{c,§}	± 14.9				D =0.54
	HC-LBP	117.6	± 8.9	118.2	± 12.8				BP = 0.001
	HC-HBP	138.0	± 13.7	127.3 ^{f,§}	± 14.7				D x BP = 0.88
	HP	125.6	± 14.7	123.0 [§]	± 13.7	125.8	± 0.5	T x D = 0.22	
	HC	124.3	± 14.4	121.2 [§]	± 14.1	125.3	± 0.6	T x BP = 0.001	
	LBP	117.5	± 8.8	118.6 ^b	± 12.0	118.1	± 0.5	T x D x BP = 0.68	
	HBP	137.8	± 13.6	128.3 [§]	± 14.8	133.0 [‡]	± 0.6		
Time	125.0	± 14.6	122.2*	± 13.9					
Resting Diastolic Blood Pressure (mmHg)	HP-LBP	76.2	± 6.4	77.6 [§]	± 8.3				T = 0.001
	HP-HBP	88.8	± 7.7	82.6 ^{c,§}	± 8.7				D = 0.004
	HC-LBP	75.1	± 6.7	75.0 ^c	± 8.8				BP = 0.001
	HC-HBP	87.9	± 8.1	81.1 ^{f,§}	± 8.6				D x BP = 0.52
	HP	81.3	± 9.3	79.6 [§]	± 8.8	81.3 [†]	± 0.3	T x D = 0.17	
	HC	79.3	± 9.4	77.0 ^{a,§}	± 9.2	79.8	± 0.4	T x BP = 0.001	
	LBP	75.7	± 6.5	76.4 ^b	± 8.6	76.0	± 0.3	T x D x BP = 0.59	
	HBP	88.5	± 7.9	82.0 [§]	± 8.7	85.1 [‡]	± 0.4		
Time	80.4	± 9.4	78.4*	± 9.1					
HP = higher protein, HC = higher carbohydrate, LBP = low blood pressure, HBP = high blood pressure, T = time effect, D = diet effect, BP = blood pressure risk factor effect, D x BP = diet by blood pressure risk factor effect, T x D = time by diet effect, T x BP = time by blood pressure risk factor effect. Values are means ± standard deviations (except group means are means ± standard error) from 223 participants in the HP-LBP group, 148 in the HP-HBP group, 196 in the HC-LBP group, 96 in the HC-HBP group, 371 in the HP total group, 292 in the HC total group, 419 in the LBP group, 244 in the HBP group, and 663 participants total. * Significantly different than baseline, p < 0.05 (univariate). † Significant diet effect, p < 0.05 (univariate). ‡ Significant blood pressure effect, p < 0.05 (univariate). ^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than LBP group, p < 0.05 (post hoc LSD). ^c Significantly different than HP-LBP group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-HBP group, p < 0.05 (post hoc LSD). ^f Significantly different than HC-LBP group, p < 0.05 (post hoc LSD). [§] Significantly different than baseline, p <0.05 (post hoc LSD).									

Table D.5: Fasting Blood Lipid Values After Ten Weeks of Exercise and Dietary Intervention Based on Blood Pressure Status

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Total Cholesterol (mmol/L)	HP-LBP	5.1	± 0.9	5.0 ^g	± 0.9				T = 0.001
	HP-HBP	5.3	± 1.0	5.0 ^g	± 1.0				D = 0.38
	HC-LBP	5.1	± 1.0	5.0 ^g	± 0.9				BP = 0.003
	HC-HBP	5.4	± 1.1	5.2 ^{f,g}	± 1.0				D x BP = 0.28
	HP	5.2	± 1.0	5.0 ^g	± 1.0	5.1	± 0.05		T x D = 0.53
	HC	5.2	± 1.0	5.0 ^g	± 1.0	5.2	± 0.06		T x BP = 0.085
	LBP	5.1	± 1.0	5.0 ^{b,g}	± 0.9	5.0	± 0.04		T x D x BP = 0.99
	HBP	5.4	± 1.0	5.1 ^g	± 1.0	5.3 [‡]	± 0.06		
	Time	5.2	± 1.0	5.0 [*]	± 1.0				
LDL (mmol/L)	HP-LBP	3.1	± 0.7	2.9 ^g	± 0.7				T = 0.001
	HP-HBP	3.2	± 0.8	3.0 ^g	± 0.8				D = 0.32
	HC-LBP	3.1	± 0.8	3.0 ^g	± 0.8				BP = 0.015
	HC-HBP	3.3	± 1.0	3.1 ^g	± 0.8				D x BP = 0.44
	HP	3.1	± 0.8	3.0 ^g	± 0.8	3.0	± 0.04		T x D = 0.66
	HC	3.1	± 0.8	3.0 ^g	± 0.8	3.1	± 0.05		T x BP = 0.38
	LBP	3.1	± 0.7	2.9 ^g	± 0.7	3.0	± 0.04		T x D x BP = 0.76
	HBP	3.2	± 0.9	3.1 ^g	± 0.8	3.1 [‡]	± 0.05		
	Time	3.1	± 0.8	3.0 [*]	± 0.8				
HDL (mmol/L)	HP-LBP	1.4	± 0.3	1.3 ^g	± 0.3				T = 0.001
	HP-HBP	1.4	± 0.4	1.3 ^g	± 0.3				D = 0.61
	HC-LBP	1.4	± 0.3	1.3 ^g	± 0.3				BP = 0.27
	HC-HBP	1.4	± 0.3	1.3 ^g	± 0.3				D x BP = 0.67
	HP	1.4	± 0.3	1.3 ^g	± 0.3	1.4	± 0.02		T x D = 0.85
	HC	1.4	± 0.3	1.3 ^g	± 0.3	1.4	± 0.02		T x BP = 0.045
	LBP	1.4	± 0.3	1.3 ^g	± 0.3	1.3	± 0.02		T x D x BP = 0.90
	HBP	1.4	± 0.3	1.3 ^g	± 0.3	1.4	± 0.02		
	Time	1.4	± 0.3	1.3 [*]	± 0.3				
Triglycerides (mmol/L)	HP-LBP	1.5	± 0.7	1.4 ^g	± 0.8				T = 0.001
	HP-HBP	1.7	± 1.0	1.6 ^{c,g}	± 0.8				D = 0.49
	HC-LBP	1.5	± 0.8	1.4	± 0.7				BP = 0.019
	HC-HBP	1.6	± 0.7	1.5	± 0.7				D x BP = 0.47
	HP	1.6	± 0.8	1.5 ^g	± 0.8	1.5	± 0.04		T x D = 0.06
	HC	1.5	± 0.7	1.5	± 0.7	1.5	± 0.05		T x BP = 0.92
	LBP	1.5	± 0.7	1.4 ^{b,g}	± 0.8	1.5	± 0.04		T x D x BP = 0.74
	HBP	1.7	± 0.9	1.6 ^g	± 0.8	1.6 [‡]	± 0.05		
	Time	1.6	± 0.8	1.5 [*]	± 0.8				
TC/HDL ratio	HP-LBP	3.9	± 0.9	3.9	± 0.9				T = 0.011
	HP-HBP	3.9	± 0.9	4.0	± 1.0				D = 0.81
	HC-LBP	3.8	± 1.0	3.9	± 1.0				BP = 0.08
	HC-HBP	4.0	± 1.1	4.1 ^g	± 1.2				D x BP = 0.61
	HP	3.9	± 0.9	3.9	± 0.9	3.9	± 0.05		T x D = 0.39
	HC	3.9	± 1.0	4.0 ^g	± 1.1	4.0	± 0.06		T x BP = 0.12
	LBP	3.9	± 0.9	3.9 ^b	± 0.9	3.9	± 0.04		T x D x BP = 0.89
	HBP	4.0	± 1.0	4.1 ^g	± 1.0	4.0	± 0.06		
	Time	3.9	± 0.9	4.0 [*]	± 1.0				

HP = higher protein, HC = higher carbohydrate, LBP = low blood pressure, HBP = high blood pressure, T = time effect, D = diet effect, BP = blood pressure risk factor effect, D x BP = diet by blood pressure risk factor effect, T x D = time by diet effect, T x BP = time by blood pressure risk factor effect, T x D x BP = time by diet by blood pressure risk factor effect.

Values are means ± standard deviations (except group means are means ± standard error) from 223 participants in the HP-LBP group, 148 in the HP-HBP group, 196 in the HC-LBP group, 96 in the HC-HBP group, 371 in the HP total group, 292 in the HC total group, 419 in the LBP group, 244 in the HBP group, and 663 participants total.

* Significantly different than baseline, $p < 0.05$ (univariate). † Significant diet effect, $p < 0.05$ (univariate). ‡ Significant blood pressure effect, $p < 0.05$ (univariate). ^a Significantly different than HP group, $p < 0.05$ (post hoc LSD). ^b Significantly different than LBP group, $p < 0.05$ (post hoc LSD). ^c Significantly different than HP-LBP group, $p < 0.05$ (post hoc LSD). ^d Significantly different than HP-HBP group, $p < 0.05$ (post hoc LSD). ^e Significantly different than HC-LBP group, $p < 0.05$ (post hoc LSD). ^g Significantly different than baseline, $p < 0.05$ (post hoc LSD).

Table D.6: Fasting Glucose and Insulin Values After Ten Weeks of Exercise and Dietary Intervention Based on Blood Pressure Status

Variable	Group	Baseline			10 Weeks			Group (SEM)			P-level
Glucose (mmol/L)	HP-LBP	5.6	±	1.1	5.4	±	0.8				T = 0.014
	HP-HBP	5.9	±	1.4	5.8 ^c	±	1.6				D = 0.15
	HC-LBP	5.6	±	1.5	5.6	±	1.1				BP = 0.08
	HC-HBP	5.6	±	0.6	5.5 ^d	±	0.7				D x BP = 0.014
	HP	5.7	±	1.2	5.6 ^e	±	1.2	5.7	±	0.06	T x D = 0.45
	HC	5.6	±	1.3	5.5	±	1.0	5.6	±	0.07	T x BP = 0.77
	LBP	5.6	±	1.3	5.5	±	1.0	5.5	±	0.05	T x D x BP = 0.92
	HBP	5.8	±	1.2	5.7	±	1.3	5.7	±	0.07	
Time	5.7	±	1.3	5.6*	±	1.1					
Insulin (uIU/mL)	HP-LBP	5.0	±	10.8	5.3	±	11.7				T = 0.47
	HP-HBP	2.6	±	4.0	3.3	±	7.2				D =0.037
	HC-LBP	9.8	±	14.3	9.5 ^c	±	11.7				BP = 0.019
	HC-HBP	5.8	±	9.8	6.8	±	12.3				D x BP = 0.001
	HP	4.0	±	8.8	4.5	±	10.1	4.0†	±	0.80	T x D = 0.87
	HC	8.3	±	12.8	8.4 ^a	±	12.0	8.0	±	0.98	T x BP = 0.52
	LBP	7.0	±	12.6	7.1	±	11.8	7.4	±	0.80	T x D x BP =0.68
	HBP	3.9	±	7.0	4.7	±	9.7	4.6‡	±	0.98	
	Time	5.7	±	10.8	6.1	±	11.1				
Calculated HOMA	HP-LBP	1.3	±	2.8	1.4	±	3.1				T = 0.68
	HP-HBP	0.7	±	1.2	0.9	±	2.5				D = 0.034
	HC-LBP	2.8	±	5.4	2.5 ^c	±	3.6				BP = 0.015
	HC-HBP	1.6	±	2.6	1.9	±	3.4				D x BP = 0.001
	HP	1.0	±	2.3	1.2	±	2.9	1.1†	±	0.23	T x D = 0.72
	HC	2.3	±	4.6	2.2 ^a	±	3.5	2.2	±	0.28	T x BP = 0.43
	LBP	1.9	±	4.2	1.8	±	3.3	2.0	±	0.23	T x D x BP = 0.60
	HBP	1.0	±	1.9	1.3	±	3.0	1.3‡	±	0.29	
Time	1.6	±	3.5	1.6	±	3.2					
HP = higher protein, HC = higher carbohydrate, LBP = low blood pressure, HBP = high blood pressure, T = time effect, D = diet effect, BP = blood pressure risk factor effect, D x BP = diet by blood pressure risk factor effect, T x D = time by diet effect, T x BP = time by blood pressure risk factor effect, T x D x BP = time by diet by blood pressure risk factor effect. Glucose values are means ± standard deviations (except group means are means ± standard error) from 223 participants in the HP-LBP group, 148 in the HP-HBP group, 196 in the HC-LBP group, 96 in the HC-HBP group, 371 in the HP total group, 292 in the HC total group, 419 in the LBP group, 244 in the HBP group, and 663 participants total. Insulin and HOMA values are means ± standard deviations (except group means are means ± standard error) from 88 participants in the HP-LBP group, 61 in the HP-HBP group, 63 in the HC-LBP group, 40 in the HC-HBP group, 149 in the HP total group, 103 in the HC total group, 151 in the LBP group, 101 in the HBP group, and 252 participants total. * Significantly different than baseline, p < 0.05 (univariate). † Significant diet effect, p < 0.05 (univariate). ‡ Significant blood pressure effect, p < 0.05 (univariate). ^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than LBP group, p < 0.05 (post hoc LSD). ^c Significantly different than HP-LBP group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-HBP group, p < 0.05 (post hoc LSD). ^e Significantly different than HC-LBP group, p < 0.05 (post hoc LSD). ^f Significantly different than baseline, p <0.05 (post hoc LSD).											

Table D.7: Fitness Parameters After Ten Weeks of Exercise and Dietary Intervention Based on Blood Pressure Status

Variable	Group	Baseline			10 Weeks			Group (SEM)			P-level
Bench Press Max Strength (1 RM / kg body weight)	HP-LBP	30.9	±	7.6	33.0 [§]	±	7.7				T = 0.001
	HP-HBP	31.0	±	7.7	32.9 [§]	±	7.0				D = 0.07
	HC-LBP	30.2	±	8.2	33.0 [§]	±	8.6				BP = 0.22
	HC-HBP	29.2	±	6.9	30.8 ^{d,f,g}	±	6.7				D x BP = 0.19
	HP	30.9	±	7.6	32.9 [§]	±	7.4	31.9 [†]	±	0.4	T x D = 0.67
	HC	29.9	±	7.8	32.2 [§]	±	8.1	30.8	±	0.5	T x BP = 0.10
	LBP	30.6	±	7.9	33.0 [§]	±	8.1	31.7	±	0.4	T x D x BP = 0.27
	HBP	30.4	±	7.4	32.1 [§]	±	7.0	31.0	±	0.5	
	Time	30.5	±	7.7	32.6 [*]	±	7.7				
Bench Press Lift Volume (kg)	HP-LBP	177.6	±	75.7	177.9	±	78.1				T = 0.96
	HP-HBP	184.2	±	85.4	187.9	±	84.1				D = 0.60
	HC-LBP	191.8	±	88.4	184.1	±	82.8				BP = 0.39
	HC-HBP	167.4	±	73.6	171.9	±	73.2				D x BP = 0.024
	HP	180.3	±	79.7	182.0	±	80.7	181.9	±	3.7	T x D = 0.64
	HC	183.8	±	84.5	180.1	±	79.9	178.8	±	4.5	T x BP = 0.30
	LBP	184.2	±	82.0	180.8	±	80.3	182.9	±	3.5	T x D x BP = 0.57
	HBP	177.9	±	81.4	181.9	±	80.4	177.9	±	4.7	
	Time	181.8	±	81.8	181.2	±	80.3				
Leg Press Max Strength (1 RM / kg body weight)	HP-LBP	162.8	±	47.1	179.0 [§]	±	54.0				T = 0.001
	HP-HBP	166.0	±	49.5	181.6 [§]	±	49.9				D = 0.047
	HC-LBP	160.6	±	51.2	176.4 [§]	±	55.3				BP = 0.46
	HC-HBP	153.8	±	50.0	164.7 ^{d,g}	±	54.4				D x BP = 0.16
	HP	164.1	±	48.1	180.1 [§]	±	52.3	172.4 [†]	±	2.7	T x D = 0.30
	HC	158.4	±	50.8	172.5 ^{a,g}	±	55.2	163.9	±	3.3	T x BP = 0.26
	LBP	161.8	±	49.0	177.8	±	54.6	169.7	±	2.6	T x D x BP = 0.40
	HBP	161.4	±	49.9	175.2	±	52.2	166.5	±	3.4	
	Time	161.7	±	49.3	176.8 [*]	±	53.6				
Leg Press Lift Volume (kg)	HP-LBP	1773.0	±	938.3	1870.5	±	1021.5				T = 0.30
	HP-HBP	1891.4	±	1008.9	2022.5	±	1151.5				D = 0.001
	HC-LBP	1696.4	±	903.1	1704.7	±	885.6				BP = 0.98
	HC-HBP	1607.6	±	780.6	1530.0 ^d	±	928.2				D x BP = 0.07
	HP	1821.8	±	968.3	1933.1 [§]	±	1077.9	1889.3 [†]	±	47.2	T x D = 0.054
	HC	1667.3	±	864.3	1647.4 ^a	±	901.7	1634.7	±	57.0	T x BP = 0.74
	LBP	1737.6	±	921.8	1793.8 [§]	±	963.4	1761.2	±	44.5	T x D x BP = 0.44
	HBP	1785.0	±	938.2	1837.8 [§]	±	1097.4	1762.9	±	59.1	
	Time	1755.4	±	927.5	1810.3	±	1015.1				
Peak VO₂ (mL/kg/min)	HP-LBP	20.5	±	4.6	22.4 [§]	±	4.6				T = 0.001
	HP-HBP	19.3	±	4.3	21.3 ^{c,g}	±	4.3				D = 0.043
	HC-LBP	20.8	±	4.4	23.6 ^{c,g}	±	5.0				BP = 0.001
	HC-HBP	19.8	±	3.7	22.0 ^{f,g}	±	4.0				D x BP = 0.85
	HP	20.0	±	4.5	21.9 [§]	±	4.5	20.9 [†]	±	0.22	T x D = 0.054
	HC	20.5	±	4.2	23.1 ^{a,g}	±	4.7	21.6	±	0.26	T x BP = 0.40
	LBP	20.7	±	4.5	22.9 ^{b,g}	±	4.8	21.8	±	0.20	T x D x BP = 0.13
	HBP	19.5	±	4.1	21.6 ^g	±	4.2	20.6 [‡]	±	0.27	
	Time	20.2	±	4.4	22.4 [*]	±	4.6				

HP = higher protein, HC = higher carbohydrate, LBP = low blood pressure, HBP = high blood pressure, T = time effect, D = diet effect, BP = blood pressure risk factor effect, D x BP = diet by blood pressure risk factor effect, T x D = time by diet effect, T x BP = time by blood pressure risk factor effect, T x D x BP = time by diet by blood pressure risk factor effect.

Strength values are means ± standard deviations (except group means are means ± standard error) from 200 participants in the HP-LBP group, 140 in the HP-HBP group, 172 in the HC-LBP group, 84 in the HC-HBP group, 340 in the HP total group, 256 in the HC total group, 372 in the LBP group, 224 in the HBP group, and 596 participants total. Peak VO₂ values are means ± standard deviations (except group means are means ± standard error) from 223 participants in the HP-LBP group, 148 in the HP-HBP group, 196 in the HC-LBP group, 96 in the HC-HBP group, 371 in the HP total group, 292 in the HC total group, 419 in the LBP group, 244 in the HBP group, and 663 participants total.

* Significantly different than baseline, $p < 0.05$ (univariate). † Significant diet effect, $p < 0.05$ (univariate). ‡ Significant blood pressure effect, $p < 0.05$ (univariate). ^a Significantly different than HP group, $p < 0.05$ (post hoc LSD). ^b Significantly different than LBP group, $p < 0.05$ (post hoc LSD).

^c Significantly different than HP-LBP group, $p < 0.05$ (post hoc LSD). ^d Significantly different than HP-HBP group, $p < 0.05$ (post hoc LSD).

^f Significantly different than HC-LBP group, $p < 0.05$ (post hoc LSD). ^g Significantly different than baseline, $p < 0.05$ (post hoc LSD).

Table D.8: Changes in SF36 - Quality of Life Inventory Values After Ten Weeks of Exercise and Dietary Intervention Based on Blood Pressure Status

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Physical Functioning	HP-LBP	77.8	± 29.3	84.8 ^a	± 25.8				T = 0.001
	HP-HBP	71.4	± 30.4	74.1 ^c	± 21.0				D = 0.60
	HC-LBP	78.7	± 21.6	84.1 ^b	± 21.4				BP = 0.008
	HC-HBP	71.0	± 19.5	79.9 ^b	± 17.1				D x BP = 0.64
	HP	75.7	± 29.7	81.3 ^b	± 24.7	77.0	± 1.85		T x D = 0.43
	HC	76.4	± 21.2	82.8 ^b	± 20.2	78.4	± 1.99		T x BP = 0.89
	LBP	78.2	± 25.8	84.5 ^{b,g}	± 23.7	81.3	± 1.53		T x D x BP = 0.17
	HBP	71.2	± 25.9	76.7 ^b	± 19.4	74.1 [‡]	± 2.24		
	Time	76.0	± 26.0	82.0 [*]	± 22.7				
Role Physical	HP-LBP	144.8	± 137.2	151.6	± 150.8				T = 0.89
	HP-HBP	136.9	± 139.4	136.0	± 143.1				D = 0.001
	HC-LBP	244.9	± 145.5	257.2 ^c	± 147.7				BP = 0.70
	HC-HBP	257.0	± 136.2	241.3 ^d	± 146.8				D x BP = 0.78
	HP	142.2	± 137.5	146.4	± 148.0	142.3 [†]	± 12.09		T x D = 0.59
	HC	248.6	± 142.3	252.3 ^a	± 147.1	250.1	± 12.97		T x BP = 0.040
	LBP	193.1	± 149.6	202.5 ^b	± 158.0	199.6	± 9.98		T x D x BP = 0.24
	HBP	191.7	± 149.8	184.0	± 153.4	192.8	± 14.65		
	Time	192.6	± 149.4	196.6	± 156.5				
Bodily Pain	HP-LBP	61.3	± 18.5	65.2 ^b	± 19.4				T = 0.014
	HP-HBP	54.6	± 19.2	58.9	± 22.8				D = 0.94
	HC-LBP	65.9	± 20.9	67.2	± 21.3				BP = 0.001
	HC-HBP	53.2	± 20.2	54.4 ^f	± 20.0				D x BP = 0.18
	HP	59.1	± 19.0	63.1 ^b	± 20.7	60.0	± 1.58		T x D = 0.20
	HC	62.0	± 21.4	63.3	± 21.6	60.2	± 1.69		T x BP = 0.98
	LBP	63.5	± 19.8	66.2 ^{b,g}	± 20.3	64.9	± 1.30		T x D x BP = 0.93
	HBP	54.0	± 19.6	56.8	± 21.5	55.3 [‡]	± 1.91		
	Time	60.5	± 20.2	63.2 [*]	± 21.1				
General Health	HP-LBP	51.3	± 28.6	55.8 ^b	± 28.9				T = 0.001
	HP-HBP	48.3	± 28.7	52.6 ^b	± 28.2				D = 0.001
	HC-LBP	65.6	± 22.1	68.5 ^{c,g}	± 21.7				BP = 0.50
	HC-HBP	63.9	± 23.0	68.0 ^{d,g}	± 23.3				D x BP = 0.76
	HP	50.4	± 28.6	54.8 ^b	± 28.6	52.0 [†]	± 2.17		T x D = 0.57
	HC	65.1	± 22.3	68.4 ^{a,g}	± 22.1	66.5	± 2.33		T x BP = 0.73
	LBP	58.2	± 26.6	62.0 ^b	± 26.4	60.3	± 1.79		T x D x BP = 0.62
	HBP	55.4	± 27.3	59.6 ^b	± 27.0	58.2	± 2.63		
	Time	57.3	± 26.8	61.2 [*]	± 26.6				
Vital	HP-LBP	40.1	± 41.2	49.8 ^b	± 43.8				T = 0.001
	HP-HBP	43.7	± 55.1	47.7 ^b	± 54.8				D = 0.10
	HC-LBP	51.0	± 18.5	55.8 ^b	± 18.0				BP = 0.94
	HC-HBP	49.0	± 18.6	54.9 ^b	± 20.0				D x BP = 0.81
	HP	41.3	± 46.1	49.1 ^b	± 47.5	45.3	± 3.08		T x D = 0.41
	HC	50.4	± 18.5	55.5 ^b	± 18.6	52.7	± 3.30		T x BP = 0.21
	LBP	45.3	± 32.7	52.7 ^b	± 33.9	49.2	± 2.54		T x D x BP = 0.07
	HBP	46.1	± 42.4	51.0 ^b	± 42.6	48.8	± 3.73		
	Time	45.6	± 36.0	52.2 [*]	± 36.8				

Table D.8: Continued

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Social Functioning	HP-LBP	45.1	± 24.3	50.1 [§]	± 25.2				T = 0.06
	HP-HBP	48.6	± 28.6	52.0	± 29.8				D = 0.001
	HC-LBP	55.1	± 22.6	57.3 ^c	± 21.7				BP = 0.019
	HC-HBP	68.7	± 27.3	66.5 ^{d,f}	± 29.1				D x BP = 0.15
	HP	46.2	± 25.7	50.7 [§]	± 26.7	48.9 [†]	± 2.06	T x D = 0.06	
	HC	59.3	± 24.9	60.1 ^a	± 24.5	61.9	± 2.21	T x BP = 0.17	
	LBP	49.9	± 24.0	53.5 [§]	± 23.8	51.9	± 1.70	T x D x BP = 0.51	
	HBP	57.8	± 29.7	58.6	± 30.2	59.0 [‡]	± 2.49		
Time	52.4	± 26.1	55.2 [*]	± 26.1					
Role Emotional	HP-LBP	220.1	± 130.2	240.4	± 143.0				T = 0.63
	HP-HBP	214.5	± 127.0	235.0	± 136.8				D = 0.001
	HC-LBP	297.6	± 215.6	292.5 ^c	± 134.4				BP = 0.79
	HC-HBP	300.8	± 116.7	282.1	± 134.2				D x BP = 0.96
	HP	218.3	± 128.7	238.6	± 140.6	227.5 [†]	± 11.51	T x D = 0.07	
	HC	298.6	± 190.3	289.4 ^a	± 133.9	293.3	± 12.36	T x BP = 0.70	
	LBP	257.5	± 180.4	265.5	± 141.0	262.6	± 9.51	T x D x BP = 0.69	
	HBP	253.9	± 129.2	256.5	± 136.9	258.1	± 13.96		
Time	256.3	± 165.6	262.7	± 139.6					
Mental Health	HP-LBP	57.8	± 15.4	66.8 [§]	± 15.5				T = 0.001
	HP-HBP	53.9	± 15.6	61.7 ^{c,§}	± 14.4				D = 0.008
	HC-LBP	59.8	± 14.7	67.2 [§]	± 12.7				BP = 0.33
	HC-HBP	61.3	± 13.8	68.7 ^{d,§}	± 10.8				D x BP = 0.06
	HP	56.5	± 15.5	65.1 [§]	± 15.3	60.1 [†]	± 1.06	T x D = 0.60	
	HC	60.3	± 14.4	67.6 ^{a,§}	± 12.1	64.2	± 1.14	T x BP = 0.76	
	LBP	58.8	± 15.1	67.0 [§]	± 14.2	62.9	± 0.88	T x D x BP = 0.72	
	HBP	57.3	± 15.2	64.9 [§]	± 13.3	61.4	± 1.29		
Time	58.3	± 15.1	66.3 [*]	± 13.9					
HP = higher protein, HC = higher carbohydrate, LBP = low blood pressure, HBP = high blood pressure, T = time effect, D = diet effect, BP = blood pressure risk factor effect, D x BP = diet by blood pressure risk factor effect, T x D = time by diet effect, T x BP = time by blood pressure risk factor effect, T x D x BP = time by diet by blood pressure risk factor effect. Values are means ± standard deviations (except group means are means ± standard error) from 102 participants in the HP-LBP group, 50 in the HP-HBP group, 95 in the HC-LBP group, 42 in the HC-HBP group, 152 in the HP total group, 137 in the HC total group, 197 in the LBP group, 92 in the HBP group, and 289 participants total. * Significantly different than baseline, p < 0.05 (univariate). † Significant diet effect, p < 0.05 (univariate). ‡ Significant blood pressure effect, p < 0.05 (univariate). ^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than LBP group, p < 0.05 (post hoc LSD). ^c Significantly different than HP-LBP group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-HBP group, p < 0.05 (post hoc LSD). ^e Significantly different than HC-LBP group, p < 0.05 (post hoc LSD). ^f Significantly different than baseline, p < 0.05 (post hoc LSD).									

Table D.9: Changes in Body Image Evaluation After Ten Weeks of Exercise and Dietary Intervention Based on Blood Pressure Status

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Appearance Evaluation	HP-LBP	2.4	± 0.7	2.8 ^g	± 0.7				T = 0.001
	HP-HBP	2.4	± 0.6	2.7 ^g	± 0.6				D = 0.58
	HC-LBP	2.5	± 0.7	2.8 ^g	± 0.7				BP = 0.80
	HC-HBP	2.4	± 0.6	2.8 ^g	± 0.7				D x BP = 0.49
	HP	2.4	± 0.6	2.7 ^g	± 0.7	2.6	± 0.04		T x D = 0.48
	HC	2.4	± 0.6	2.8 ^g	± 0.7	2.6	± 0.04		T x BP = 0.57
	LBP	2.4	± 0.7	2.8 ^g	± 0.7	2.6	± 0.04		T x D x BP = 0.50
	HBP	2.4	± 0.6	2.8 ^g	± 0.6	2.6	± 0.05		
	Time	2.4	± 0.6	2.8*	± 0.7				
Appearance Orientation	HP-LBP	4.1	± 0.8	4.1	± 0.9				T = 0.32
	HP-HBP	4.2	± 0.9	4.2	± 1.0				D = 0.043
	HC-LBP	3.9	± 0.7	3.8 ^c	± 0.8				BP = 0.007
	HC-HBP	4.1	± 0.9	4.1 ^f	± 0.9				D x BP = 0.34
	HP	4.1	± 0.9	4.1	± 0.9	4.1 [†]	± 0.05		T x D = 0.55
	HC	3.9	± 0.8	3.9 ^a	± 0.9	4.0	± 0.06		T x BP = 0.68
	LBP	4.0	± 0.8	3.9 ^b	± 0.8	3.9	± 0.05		T x D x BP = 0.53
	HBP	4.2	± 0.9	4.2	± 0.9	4.2 [‡]	± 0.07		
	Time	4.1	± 0.8	4.0	± 0.9				
Body Area Satisfaction	HP-LBP	2.1	± 0.7	2.3 ^g	± 0.8				T = 0.001
	HP-HBP	1.9	± 0.8	2.2 ^g	± 0.9				D = 0.007
	HC-LBP	2.3	± 0.7	2.7 ^{c,g}	± 0.8				BP = 0.001
	HC-HBP	2.0	± 0.7	2.3 ^{f,g}	± 0.8				D x BP = 0.23
	HP	2.0	± 0.8	2.3 ^g	± 0.8	2.1 [†]	± 0.05		T x D = 0.31
	HC	2.2	± 0.7	2.5 ^{a,g}	± 0.8	2.3	± 0.06		T x BP = 0.52
	LBP	2.2	± 0.7	2.5 ^{b,g}	± 0.8	2.4	± 0.05		T x D x BP = 0.25
	HBP	2.0	± 0.7	2.3 ^g	± 0.9	2.1 [‡]	± 0.06		
	Time	2.1	± 0.7	2.4*	± 0.8				
Overweight Preoccupation	HP-LBP	2.8	± 0.7	3.4 ^g	± 0.7				T = 0.001
	HP-HBP	2.9	± 0.6	3.4 ^g	± 0.8				D = 0.33
	HC-LBP	2.8	± 0.8	3.3 ^g	± 0.7				BP = 0.51
	HC-HBP	2.9	± 0.7	3.3 ^g	± 0.8				D x BP = 0.85
	HP	2.9	± 0.7	3.4 ^g	± 0.7	3.1	± 0.04		T x D = 0.11
	HC	2.9	± 0.7	3.3 ^g	± 0.7	3.1	± 0.05		T x BP = 0.49
	LBP	2.8	± 0.7	3.3 ^g	± 0.7	3.1	± 0.04		T x D x BP = 0.61
	HBP	2.9	± 0.7	3.4 ^g	± 0.8	3.1	± 0.05		
	Time	2.9	± 0.7	3.3*	± 0.7				
Self-Classified Weight	HP-LBP	4.3	± 0.7	4.2	± 0.5				T = 0.013
	HP-HBP	4.2	± 0.8	4.1	± 0.8				D = 0.12
	HC-LBP	4.2	± 0.7	4.0	± 0.7				BP = 0.57
	HC-HBP	4.2	± 0.7	4.1	± 0.7				D x BP = 0.29
	HP	4.2	± 0.7	4.2	± 0.7	4.2	± 0.04		T x D = 0.66
	HC	4.2	± 0.7	4.0	± 0.7	4.1	± 0.04		T x BP = 0.69
	LBP	4.2	± 0.7	4.1	± 0.6	4.2	± 0.03		T x D x BP = 0.61
	HBP	4.2	± 0.7	4.1	± 0.8	4.1	± 0.05		
	Time	4.2	± 0.7	4.1*	± 0.7				

Table D.9: Continued

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Rosenberg Self Esteem	HP-LBP	25.6	± 3.8	26.0	± 3.6				T = 0.006
	HP-HBP	25.8	± 4.2	26.8 ^a	± 4.2				D = 0.54
	HC-LBP	25.7	± 3.6	25.9	± 3.7				BP = 0.37
	HC-HBP	25.8	± 4.5	26.1	± 4.6				D x BP = 0.70
	HP	25.7	± 3.9	26.3 ^a	± 3.8	26.1	± 0.23	T x D = 0.17	
	HC	25.7	± 3.9	25.9	± 4.0	25.8	± 0.27	T x BP = 0.34	
	LBP	25.6	± 3.7	26.0	± 3.6	25.8	± 0.21	T x D x BP = 0.42	
	HBP	25.8	± 4.3	26.5 ^a	± 4.3	26.1	± 0.28		
	Time	25.7	± 3.9	26.2*	± 3.9				
Social Physique Anxiety	HP-LBP	31.5	± 6.8	31.5	± 6.9				T = 0.61
	HP-HBP	30.8	± 5.4	31.1	± 5.1				D = 0.95
	HC-LBP	31.1	± 6.1	31.1	± 6.5				BP = 0.70
	HC-HBP	31.1	± 5.8	31.5	± 5.3				D x BP = 0.45
	HP	31.3	± 6.3	31.4	± 6.2	31.2	± 0.33	T x D = 0.98	
	HC	31.1	± 6.0	31.3	± 6.1	31.2	± 0.39	T x BP = 0.61	
	LBP	31.3	± 6.5	31.3	± 6.7	31.3	± 0.31	T x D x BP = 0.84	
	HBP	30.9	± 5.5	31.3	± 5.2	31.1	± 0.40		
	Time	31.2	± 6.1	31.3	± 6.2				
HP = higher protein, HC = higher carbohydrate, LBP = low blood pressure, HBP = high blood pressure, T = time effect, D = diet effect, BP = blood pressure risk factor effect, D x BP = diet by blood pressure risk factor effect, T x D = time by diet effect, T x BP = time by blood pressure risk factor effect, T x D x BP = time by diet by blood pressure risk factor effect. Values are means ± standard deviations (except group means are means ± standard error) from 154 participants in the HP-LBP group, 102 in the HP-HBP group, 128 in the HC-LBP group, 67 in the HC-HBP group, 256 in the HP total group, 195 in the HC total group, 282 in the LBP group, 169 in the HBP group, and 451 participants total. * Significantly different than baseline, p < 0.05 (univariate). † Significant diet effect, p < 0.05 (univariate). ‡ Significant blood pressure effect, p < 0.05 (univariate). ^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than LBP group, p < 0.05 (post hoc LSD). ^c Significantly different than HP-LBP group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-HBP group, p < 0.05 (post hoc LSD). ^e Significantly different than HC-LBP group, p < 0.05 (post hoc LSD). ^f Significantly different than baseline, p <0.05 (post hoc LSD).									

APPENDIX E

GLUCOSE RISK FACTOR TABLES

Table E.1: Body Composition Before and After Ten Weeks of Exercise and Dietary Intervention Based on Glucose Status and Measured via DEXA Scan

Variable	Group	Baseline			10 Weeks			Group (SEM)			P-level
Scanned Mass (kg)	HP-LG	87.5	±	17.6	83.7 ^g	±	17.0				T = 0.001
	HP-HG	91.5	±	18.5	87.5 ^{c,g}	±	18.0				D = 0.001
	HC-LG	80.8	±	13.9	77.9 ^{c,g}	±	13.1				G = 0.002
	HC-HG	85.0	±	14.7	81.9 ^{d,f,g}	±	14.3				D x G = 0.99
	HP	89.2	±	18.1	85.3 ^g	±	17.5	87.6 [†]	±	0.84	T x D = 0.001
	HC	82.5	±	14.3	79.5 ^{a,g}	±	13.7	81.4	±	0.96	T x G = 0.47
	LG	84.5	±	16.4	81.1 ^{b,g}	±	15.6	82.5	±	0.82	T x D x G = 0.88
	HG	88.8	±	17.3	85.1 ^g	±	16.7	86.5 [‡]	±	0.98	
Time	86.2	±	16.9	82.7 [*]	±	16.2					
Fat Mass (kg)	HP-LG	40.2	±	10.9	37.1 ^g	±	10.8				T = 0.001
	HP-HG	42.4	±	12.0	39.3 ^{c,g}	±	11.6				D = 0.001
	HC-LG	36.1	±	8.7	33.9 ^{c,g}	±	8.5				G = 0.006
	HC-HG	38.6	±	9.4	35.9 ^{d,g}	±	8.9				D x G = 0.99
	HP	41.1	±	11.4	38.0 ^g	±	11.2	39.7 [†]	±	0.54	T x D = 0.004
	HC	37.1	±	9.1	34.7 ^{a,g}	±	8.7	36.1	±	0.61	T x G = 0.23
	LG	38.3	±	10.2	35.6 ^{b,g}	±	9.9	36.8	±	0.52	T x D x G =0.27
	HG	40.8	±	11.1	37.8 ^g	±	10.7	39.0 [‡]	±	0.62	
Time	39.3	±	10.7	36.5 [*]	±	10.3					
Lean Mass (kg)	HP-LG	45.6	±	7.8	44.8 ^g	±	7.4				T = 0.001
	HP-HG	47.3	±	7.5	46.4 ^{c,g}	±	7.3				D = 0.001
	HC-LG	42.9	±	6.4	42.2 ^{c,g}	±	6.0				G = 0.002
	HC-HG	44.7	±	6.4	44.2 ^{d,f,g}	±	6.4				D x G = 0.81
	HP	46.3	±	7.7	45.5 ^g	±	7.4	46.0 [†]	±	0.36	T x D = 0.12
	HC	43.6	±	6.4	43.0 ^{a,g}	±	6.2	43.5	±	0.41	T x G = 0.68
	LG	44.4	±	7.3	43.7 ^{b,g}	±	6.9	43.9	±	0.35	T x D x G = 0.23
	HG	46.2	±	7.2	45.5 ^g	±	7.0	45.6 [‡]	±	0.42	
Time	45.1	±	7.3	44.4 [*]	±	7.0					
Body Fat (%)	HP-LG	45.3	±	4.2	43.7 ^g	±	4.6				T = 0.001
	HP-HG	45.7	±	4.7	44.2 ^g	±	4.8				D = 0.06
	HC-LG	44.4	±	4.2	43.2 ^g	±	4.4				G = 0.14
	HC-HG	45.2	±	4.1	43.5 ^g	±	4.1				D x G = 0.84
	HP	45.5	±	4.4	43.9 ^g	±	4.7	44.8	±	0.23	T x D = 0.28
	HC	44.7	±	4.2	43.4 ^g	±	4.3	44.1	±	0.26	T x G = 0.18
	LG	44.9	±	4.2	43.5 ^g	±	4.5	44.2	±	0.22	T x D x G = 0.06
	HG	45.5	±	4.4	43.9 ^g	±	4.5	44.7	±	0.26	
Time	45.2	±	4.3	43.7 [*]	±	4.5					
HP = higher protein, HC = higher carbohydrate, LG = low glucose, HG = high glucose, T = time effect, D = diet effect, G = glucose risk factor effect, D x G = diet by glucose risk factor effect, T x D = time by diet effect, T x G = time by glucose risk factor effect, T x D x G = time by diet by glucose risk factor effect. Values are means ± standard deviations (except group means are means ± standard error) from 214 participants in the HP-LG group, 157 in the HP-HG group, 176 in the HC-LG group, 116 in the HC-HG group, 371 in the HP total group, 292 in the HC total group, 390 in the LG group, 273 in the HG group, and 663 participants total. * Significantly different than baseline, p < 0.05 (univariate). † Significant diet effect, p < 0.05 (univariate). ‡ Significant glucose effect, p < 0.05 (univariate). ^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than LG group, p < 0.05 (post hoc LSD). ^c Significantly different than HP-LG group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-HG group, p < 0.05 (post hoc LSD). ^e Significantly different than HC-LG group, p < 0.05 (post hoc LSD). ^f Significantly different than baseline, p < 0.05 (post hoc LSD).											

Table E.2: Body Composition Before and After Ten Weeks of Exercise and Dietary Intervention Based on Glucose Status and Measured via Anthropometric Measurements

Variable	Grou p	Baseline		10 Weeks		Group (SEM)			P-level
Weight (kg)	HP-LG	94.0	± 18.5	89.7 ^g	± 17.8				T = 0.001
	HP-HG	98.2	± 19.6	93.9 ^{c,g}	± 18.9				D = 0.001
	HC-LG	86.7	± 14.3	83.7 ^{c,g}	± 13.4				G = 0.001
	HC-HG	91.8	± 15.4	88.3 ^{d,f,g}	± 14.9				D x G = 0.82
	HP	95.8	± 19.1	91.5 ^g	± 18.3	94.0 [†]	± 0.9	T x D = 0.001	
	HC	88.8	± 14.9	85.5 ^{a,g}	± 14.2	87.6	± 1.0	T x G = 0.32	
	LG	90.7	± 17.1	87.0 ^{b,g}	± 16.2	88.5	± 0.9	T x D x G = 0.44	
	HG	95.5	± 18.2	91.5 ^g	± 17.5	93.1 [‡]	± 1.0		
Time	92.7	± 17.7	88.9 [*]	± 16.9					
Body Mass Index (kg/m ²)	HP-LG	35.1	± 6.4	33.5 ^g	± 6.2				T = 0.001
	HP-HG	37.0	± 6.7	35.4 ^{c,g}	± 6.5				D = 0.001
	HC-LG	32.7	± 5.1	31.5 ^{c,g}	± 4.8				G = 0.001
	HC-HG	34.3	± 5.3	32.9 ^{d,f,g}	± 5.2				D x G = 0.68
	HP	35.9	± 6.6	34.3 ^g	± 6.4	35.3 [†]	± 0.3	T x D = 0.001	
	HC	33.3	± 5.2	32.1 ^{a,g}	± 5.0	32.8	± 0.3	T x G = 0.32	
	LG	34.0	± 6.0	32.6 ^{b,g}	± 5.7	33.2	± 0.3	T x D x G = 0.47	
	HG	35.9	± 6.3	34.4 ^g	± 6.1	34.9 [‡]	± 0.4		
Time	34.8	± 6.2	33.3 [*]	± 5.9					
Waist Circumference (cm)	HP-LG	99.4	± 12.6	95.1 ^g	± 12.5				T = 0.001
	HP-HG	104.0	± 14.2	100.4 ^{c,g}	± 13.5				D = 0.001
	HC-LG	93.7	± 11.0	90.9 ^{c,g}	± 10.5				G = 0.001
	HC-HG	98.9	± 12.0	95.2 ^{d,f,g}	± 11.7				D x G = 0.92
	HP	101.3	± 13.5	97.3 ^g	± 13.2	99.7 [†]	± 0.6	T x D = 0.14	
	HC	95.8	± 11.7	92.6 ^{a,g}	± 11.2	94.7	± 0.7	T x G = 0.90	
	LG	96.8	± 12.2	93.2 ^{b,g}	± 11.8	94.8	± 0.6	T x D x G = 0.07	
	HG	101.8	± 13.5	98.2 ^g	± 13.0	99.6 [‡]	± 0.7		
Time	98.9	± 13.0	95.2 [*]	± 12.6					
Hip Circumference (cm)	HP-LG	121.7	± 13.4	118.3 ^g	± 12.8				T = 0.001
	HP-HG	124.1	± 14.5	121.3 ^{c,g}	± 14.5				D = 0.001
	HC-LG	116.7	± 11.2	114.4 ^{c,g}	± 10.6				G = 0.001
	HC-HG	120.9	± 11.6	117.5 ^{d,f,g}	± 11.0				D x G = 0.62
	HP	122.7	± 13.9	119.5 ^g	± 13.6	121.3 [†]	± 0.6	T x D = 0.38	
	HC	118.3	± 11.5	115.6 ^{a,g}	± 10.9	117.4	± 0.7	T x G = 0.63	
	LG	119.5	± 12.7	116.5 ^{b,g}	± 12.0	117.8	± 0.6	T x D x G = 0.033	
	HG	122.7	± 13.4	119.7 ^a	± 13.3	120.9 [‡]	± 0.8		
Time	120.8	± 13.1	117.8 [*]	± 12.6					
HP = higher protein, HC = higher carbohydrate, LG = low glucose, HG = high glucose, T = time effect, D = diet effect, G = glucose risk factor effect, D x G = diet by glucose risk factor effect, T x D = time by diet effect, T x G = time by glucose risk factor effect, T x D x G = time by diet by glucose risk factor effect. Values are means ± standard deviations (except group means are means ± standard error) from 214 participants in the HP-LG group, 157 in the HP-HG group, 176 in the HC-LG group, 116 in the HC-HG group, 371 in the HP total group, 292 in the HC total group, 390 in the LG group, 273 in the HG group, and 663 participants total. * Significantly different than baseline, p < 0.05 (univariate). † Significant diet effect, p < 0.05 (univariate). ‡ Significant glucose effect, p < 0.05 (univariate). ^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than LG group, p < 0.05 (post hoc LSD). ^c Significantly different than HP-LG group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-HG group, p < 0.05 (post hoc LSD). ^e Significantly different than HC-LG group, p < 0.05 (post hoc LSD). ^f Significantly different than baseline, p < 0.05 (post hoc LSD).									

Table E.3: Resting Energy Expenditure After Ten Weeks of Exercise and Dietary Intervention Based on Glucose Status

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Resting Energy Expenditure (kcal/day)	HP-LG	1657.6	± 272.7	1602.8 ^g	± 292.3				T = 0.001
	HP-HG	1753.6	± 294.0	1677.7 ^{a,g}	± 288.0				D = 0.001
	HC-LG	1565.6	± 238.9	1538.5 ^c	± 247.1				G = 0.001
	HC-HG	1656.0	± 253.1	1582.3 ^{d,g}	± 250.2				D x G = 0.65
	HP	1697.4	± 285.2	1633.8 ^e	± 292.5	1672.9 [†]	± 13.3		T x D = 0.39
	HC	1600.7	± 248.0	1555.5 ^{a,g}	± 248.8	1585.6	± 15.3		T x G = 0.051
	LG	1616.5	± 261.8	1574.0 ^{b,g}	± 274.5	1591.1	± 12.8		T x D x G = 0.46
	HG	1712.6	± 281.2	1637.6 ^a	± 276.3	1667.4 [‡]	± 15.7		
	Time	1655.2	± 273.7	1599.7 [*]	± 276.8				

HP = higher protein, HC = higher carbohydrate, LG = low glucose, HG = high glucose, T = time effect, D = diet effect, G = glucose risk factor effect, D x G = diet by glucose risk factor effect, T x D = time by diet effect, T x G = time by glucose risk factor effect, T x D x G = time by diet by glucose risk factor effect.

Values are means ± standard deviations (except group means are means ± standard error) from 209 participants in the HP-LG group, 148 in the HP-HG group, 169 in the HC-LG group, 107 in the HC-HG group, 357 in the HP total group, 276 in the HC total group, 378 in the LG group, 255 in the HG group, and 633 participants total.

* Significantly different than baseline, $p < 0.05$ (univariate). [†] Significant diet effect, $p < 0.05$ (univariate). [‡] Significant glucose effect, $p < 0.05$ (univariate). ^a Significantly different than HP group, $p < 0.05$ (post hoc LSD). ^b Significantly different than LG group, $p < 0.05$ (post hoc LSD). ^c Significantly different than HP-LG group, $p < 0.05$ (post hoc LSD). ^d Significantly different than HP-HG group, $p < 0.05$ (post hoc LSD). ^e Significantly different than HC-LG group, $p < 0.05$ (post hoc LSD). ^f Significantly different than baseline, $p < 0.05$ (post hoc LSD).

Table E.4: Resting Hemodynamic Measurements After Ten Weeks of Exercise and Dietary Intervention Based on Glucose Status

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Resting Heart Rate (bpm)	HP-LG	70.9	± 10.0	67.9 [§]	± 10.0				T = 0.001
	HP-HG	73.5	± 10.6	70.3 ^{c,§}	± 9.2				D = 0.21
	HC-LG	70.9	± 10.2	67.9 [§]	± 9.2				G = 0.019
	HC-HG	71.5	± 11.1	68.8 [§]	± 11.0				D x G = 0.20
	HP	72.0	± 10.3	68.9 [§]	± 9.7	70.6	± 0.5		T x D = 0.79
	HC	71.2	± 10.5	68.2 [§]	± 9.9	69.8	± 0.5		T x G = 1.0
	LG	70.9	± 10.1	67.9 ^{b,§}	± 9.6	69.4	± 0.4		T x D x G = 0.78
	HG	72.7	± 10.8	69.6 [§]	± 10.0	71.0 [‡]	± 0.5		
	Time	71.6	± 10.4	68.6 [*]	± 9.8				
Resting Systolic Blood Pressure (mmHg)	HP-LG	122.8	± 12.8	122.0	± 13.3				T = 0.001
	HP-HG	129.3	± 16.3	124.2 [§]	± 14.2				D = 0.13
	HC-LG	123.3	± 14.5	119.1 ^{c,§}	± 12.6				G = 0.001
	HC-HG	125.8	± 14.2	124.3 ^f	± 15.7				D x G = 0.77
	HP	125.6	± 14.7	123.0 [§]	± 13.7	124.6	± 0.6		T x D = 0.92
	HC	124.3	± 14.4	121.2 [§]	± 14.1	123.1	± 0.7		T x G = 0.49
	LG	123.0	± 13.6	120.7 ^{b,§}	± 13.1	121.8	± 0.6		T x D x G = 0.002
	HG	127.8	± 15.5	124.3 [§]	± 14.8	125.9 [‡]	± 0.7		
	Time	125.0	± 14.6	122.2 [*]	± 13.9				
Resting Diastolic Blood Pressure (mmHg)	HP-LG	80.0	± 8.3	79.3	± 8.6				T = 0.001
	HP-HG	83.0	± 10.2	80.0 [§]	± 9.1				D = 0.001
	HC-LG	78.4	± 9.8	76.1 ^{c,§}	± 9.1				G = 0.001
	HC-HG	80.8	± 8.6	78.4 ^{f,§}	± 9.2				D x G = 0.74
	HP	81.3	± 9.3	79.6 [§]	± 8.8	80.6 [†]	± 0.4		T x D = 0.55
	HC	79.3	± 9.4	77.0a,g	± 9.2	78.4	± 0.5		T x G = 0.11
	LG	79.3	± 9.0	77.9 ^{b,§}	± 8.9	78.4	± 0.4		T x D x G = 0.15
	HG	82.1	± 9.6	79.3 [§]	± 9.2	80.5 [‡]	± 0.5		
	Time	80.4	± 9.4	78.4 [*]	± 9.1				

HP = higher protein, HC = higher carbohydrate, LG = low glucose, HG = high glucose, T = time effect, D = diet effect, G = glucose risk factor effect, D x G = diet by glucose risk factor effect, T x D = time by diet effect, T x G = time by glucose risk factor effect, T x D x G = time by diet by glucose risk factor effect.

Values are means ± standard deviations (except group means are means ± standard error) from 214 participants in the HP-LG group, 157 in the HP-HG group, 176 in the HC-LG group, 116 in the HC-HG group, 371 in the HP total group, 292 in the HC total group, 390 in the LG group, 273 in the HG group, and 663 participants total.

* Significantly different than baseline, p < 0.05 (univariate). † Significant diet effect, p < 0.05 (univariate). ‡ Significant glucose effect, p < 0.05 (univariate). ^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than LG group, p < 0.05 (post hoc LSD). ^c Significantly different than HP-LG group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-HG group, p < 0.05 (post hoc LSD). ^e Significantly different than HC-LG group, p < 0.05 (post hoc LSD). ^f Significantly different than baseline, p < 0.05 (post hoc LSD).

Table E.5: Fasting Blood Lipid Values After Ten Weeks of Exercise and Dietary Intervention Based on Glucose Status

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Total Cholesterol (mmol/L)	HP-LG	5.0	± 0.9	4.9	± 0.9				T = 0.001
	HP-HG	5.4	± 1.0	5.1 ^g	± 1.0				D = 0.72
	HC-LG	5.1	± 1.0	5.0	± 0.9				G = 0.001
	HC-HG	5.4	± 1.0	5.1 ^g	± 1.0				D x G = 0.54
	HP	5.2	± 1.0	5.0 ^g	± 1.0	5.1	± 0.05		T x D = 0.47
	HC	5.2	± 1.0	5.0 ^g	± 1.0	5.1	± 0.05		T x G = 0.001
	LG	5.1	± 0.9	5.0 ^g	± 0.9	5.0	± 0.05		T x D x G = 0.77
	HG	5.4	± 1.0	5.1 ^g	± 1.0	5.2 [‡]	± 0.05		
	Time	5.2	± 1.0	5.0*	± 1.0				
LDL (mmol/L)	HP-LG	2.9	± 0.6	2.9	± 0.7				T = 0.001
	HP-HG	3.3	± 0.8	3.1 ^{c,g}	± 0.8				D = 0.55
	HC-LG	3.0	± 0.8	3.0	± 0.8				G = 0.001
	HC-HG	3.3	± 0.9	3.1 ^g	± 0.8				D x G = 0.41
	HP	3.1	± 0.8	3.0 ^g	± 0.8	3.1	± 0.04		T x D = 0.66
	HC	3.1	± 0.8	3.0 ^g	± 0.8	3.1	± 0.04		T x G = 0.001
	LG	3.0	± 0.7	2.9 ^{b,g}	± 0.7	3.0	± 0.04		T x D x G = 0.57
	HG	3.3	± 0.8	3.1 ^g	± 0.8	3.2 [‡]	± 0.04		
	Time	3.1	± 0.8	3.0*	± 0.8				
HDL (mmol/L)	HP-LG	1.4	± 0.4	1.4 ^g	± 0.3				T = 0.001
	HP-HG	1.3	± 0.3	1.2 ^{c,g}	± 0.3				D = 0.72
	HC-LG	1.4	± 0.3	1.4 ^g	± 0.3				G = 0.001
	HC-HG	1.4	± 0.3	1.3 ^{f,g}	± 0.3				D x G = 0.48
	HP	1.4	± 0.3	1.3 ^g	± 0.3	1.3	± 0.02		T x D = 0.96
	HC	1.4	± 0.3	1.3 ^g	± 0.3	1.3	± 0.02		T x G = 0.011
	LG	1.4	± 0.4	1.4 ^{b,g}	± 0.3	1.4	± 0.02		T x D x G = 0.27
	HG	1.3	± 0.3	1.2 ^g	± 0.3	1.3 [‡]	± 0.02		
	Time	1.4	± 0.3	1.3*	± 0.3				
Triglycerides (mmol/L)	HP-LG	1.4	± 0.8	1.3 ^g	± 0.7				T = 0.001
	HP-HG	1.8	± 0.9	1.7 ^{c,g}	± 0.9				D = 0.45
	HC-LG	1.4	± 0.6	1.4	± 0.7				G = 0.001
	HC-HG	1.7	± 0.8	1.6 ^{f,g}	± 0.8				D x G = 0.21
	HP	1.6	± 0.8	1.5 ^g	± 0.8	1.6	± 0.04		T x D = 0.09
	HC	1.5	± 0.7	1.5	± 0.7	1.5	± 0.04		T x G = 0.09
	LG	1.4	± 0.7	1.3 ^{b,g}	± 0.7	1.4	± 0.04		T x D x G = 0.14
	HG	1.8	± 0.9	1.7 ^g	± 0.9	1.7 [‡]	± 0.04		
	Time	1.6	± 0.8	1.5*	± 0.8				
TC/HDL ratio	HP-LG	3.7	± 0.8	3.7	± 0.8				T = 0.025
	HP-HG	4.2	± 0.9	4.3 ^c	± 1.0				D = 0.93
	HC-LG	3.8	± 1.0	3.8	± 1.0				G = 0.001
	HC-HG	4.1	± 1.0	4.2 ^f	± 1.1				D x G = 0.20
	HP	3.9	± 0.9	3.9	± 0.9	4.0	± 0.05		T x D = 0.42
	HC	3.9	± 1.0	4.0 ^g	± 1.1	4.0	± 0.05		T x G = 0.70
	LG	3.7	± 0.9	3.8 ^b	± 0.9	3.7	± 0.05		T x D x G = 0.68
	HG	4.2	± 1.0	4.2	± 1.1	4.2 [‡]	± 0.05		
	Time	3.9	± 0.9	4.0*	± 1.0				

HP = higher protein, HC = higher carbohydrate, LG = low glucose, HG = high glucose, T = time effect, D = diet effect, G = glucose risk factor effect, D x G = diet by glucose risk factor effect, T x D = time by diet effect, T x G = time by glucose risk factor effect, T x D x G = time by diet by glucose risk factor effect.

Values are means ± standard deviations (except group means are means ± standard error) from 214 participants in the HP-LG group, 157 in the HP-HG group, 176 in the HC-LG group, 116 in the HC-HG group, 371 in the HP total group, 292 in the HC total group, 390 in the LG group, 273 in the HG group, and 663 participants total.

* Significantly different than baseline, $p < 0.05$ (univariate). † Significant diet effect, $p < 0.05$ (univariate). ‡ Significant glucose effect, $p < 0.05$ (univariate). ^a Significantly different than HP group, $p < 0.05$ (post hoc LSD). ^b Significantly different than LG group, $p < 0.05$ (post hoc LSD). ^c Significantly different than HP-LG group, $p < 0.05$ (post hoc LSD). ^d Significantly different than HP-HG group, $p < 0.05$ (post hoc LSD). ^e Significantly different than HC-LG group, $p < 0.05$ (post hoc LSD). ^f Significantly different than baseline, $p < 0.05$ (post hoc LSD).

Table E.6: Fasting Glucose and Insulin Values After Ten Weeks of Exercise and Dietary Intervention Based on Glucose Status

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Glucose (mmol/L)	HP-LG	5.1	± 0.4	5.1	± 0.5				T = 0.001
	HP-HG	6.6	± 1.5	6.2 ^{c,g}	± 1.5				D = 0.32
	HC-LG	5.1	± 0.4	5.3 ^g	± 0.5				G = 0.001
	HC-HG	6.4	± 1.7	5.9 ^{d,f,g}	± 1.4				D x G = 0.07
	HP	5.7	± 1.2	5.6 ^g	± 1.2	5.7	± 0.05		T x D = 0.81
	HC	5.6	± 1.3	5.5 ^g	± 1.0	5.7	± 0.06		T x G = 0.001
	LG	5.1	± 0.4	5.2 ^{b,g}	± 0.5	5.1	± 0.05		T x D x G = 0.017
	HG	6.5	± 1.6	6.1 ^b	± 1.5	6.3 [‡]	± 0.06		
	Time	5.7	± 1.3	5.6 [*]	± 1.1				
Insulin (uIU/mL)	HP-LG	2.9	± 7.7	3.4	± 8.9				T = 0.74
	HP-HG	6.9	± 10.8	7.6 ^c	± 12.4				D = 0.002
	HC-LG	5.6	± 6.8	6.6	± 8.2				G = 0.001
	HC-HG	13.1	± 18.8	11.9 ^f	± 16.5				D x G = 0.38
	HP	4.0	± 8.8	4.5	± 10.1	5.2 [†]	± 0.87		T x D = 0.59
	HC	8.3	± 12.8	8.4 ^a	± 12.0	9.3	± 0.97		T x G = 0.45
	LG	3.9	± 7.5	4.6 ^b	± 8.7	4.6	± 0.73		T x D x G = 0.36
	HG	9.9	± 15.4	9.7	± 14.6	9.9 [‡]	± 1.08		
	Time	5.7	± 10.8	6.1	± 11.1				
Calculated HOMA	HP-LG	0.7	± 1.9	0.8	± 2.3				T = 0.94
	HP-HG	2.0	± 3.1	2.2 ^c	± 3.9				D = 0.002
	HC-LG	1.3	± 1.6	1.6	± 2.1				G = 0.001
	HC-HG	4.2	± 7.1	3.4 ^f	± 5.1				D x G = 0.19
	HP	1.0	± 2.3	1.2	± 2.9	1.4 [†]	± 0.25		T x D = 0.38
	HC	2.3	± 4.6	2.2 ^a	± 3.5	2.6	± 0.28		T x G = 0.28
	LG	0.9	± 1.8	1.1 ^b	± 2.3	1.1	± 0.21		T x D x G = 0.19
	HG	3.0	± 5.5	2.8	± 4.5	2.9 [‡]	± 0.31		
	Time	1.6	± 3.5	1.6	± 3.2				

HP = higher protein, HC = higher carbohydrate, LG = low glucose, HG = high glucose, T = time effect, D = diet effect, G = glucose risk factor effect, D x G = diet by glucose risk factor effect, T x D = time by diet effect, T x G = time by glucose risk factor effect, T x D x G = time by diet by glucose risk factor effect.

Glucose values are means ± standard deviations (except group means are means ± standard error) from 214 participants in the HP-LG group, 157 in the HP-HG group, 176 in the HC-LG group, 116 in the HC-HG group, 371 in the HP total group, 292 in the HC total group, 390 in the LG group, 273 in the HG group, and 663 participants total. Insulin and HOMA values are means ± standard deviations (except group means are means ± standard error) from 109 participants in the HP-LG group, 40 in the HP-HG group, 67 in the HC-LG group, 36 in the HC-HG group, 149 in the HP total group, 103 in the HC total group, 176 in the LG group, 76 in the HG group, and 252 participants total.

* Significantly different than baseline, $p < 0.05$ (univariate). † Significant diet effect, $p < 0.05$ (univariate). ‡ Significant glucose effect, $p < 0.05$ (univariate). ^a Significantly different than HP group, $p < 0.05$ (post hoc LSD). ^b Significantly different than LG group, $p < 0.05$ (post hoc LSD). ^c Significantly different than HP-LG group, $p < 0.05$ (post hoc LSD). ^d Significantly different than HP-HG group, $p < 0.05$ (post hoc LSD). ^e Significantly different than HC-LG group, $p < 0.05$ (post hoc LSD). ^f Significantly different than baseline, $p < 0.05$ (post hoc LSD).

Table E.7: Fitness Parameters After Ten Weeks of Exercise and Dietary Intervention Based on Glucose Status

Variable	Group	Baseline			10 Weeks			Group (SEM)			P-level
Bench Press Max Strength (1 RM / kg body weight)	HP-LG	31.3	±	7.7	33.4 ^g	±	7.6				T = 0.001
	HP-HG	30.5	±	7.4	32.3 ^g	±	7.1				D = 0.15
	HC-LG	30.1	±	8.2	32.7 ^g	±	8.4				G = 0.13
	HC-HG	29.5	±	7.2	31.6 ^g	±	7.6				D x G = 0.96
	HP	30.9	±	7.6	32.9 ^g	±	7.4	31.9	±	0.4	T x D = 0.41
	HC	29.9	±	7.8	32.2 ^g	±	8.1	31.0	±	0.5	T x G = 0.34
	LG	30.8	±	8.0	33.1 ^g	±	8.0	31.9	±	0.4	T x D x G = 0.88
	HG	30.1	±	7.3	32.0 ^g	±	7.3	30.9	±	0.5	
	Time	30.5	±	7.7	32.6 [*]	±	7.7				
Bench Press Lift Volume (kg)	HP-LG	177.4	±	79.5	182.3	±	81.0				T = 0.76
	HP-HG	184.3	±	80.1	181.5	±	80.5				D = 0.82
	HC-LG	181.7	±	81.0	176.6	±	87.4				G = 0.38
	HC-HG	187.0	±	89.9	185.4	±	67.0				D x G = 0.73
	HP	180.3	±	79.7	182.0	±	80.7	181.4	±	3.7	T x D = 0.55
	HC	183.8	±	84.5	180.1	±	79.9	182.7	±	4.4	T x G = 0.78
	LG	179.3	±	80.1	179.8	±	83.8	179.5	±	3.7	T x D x G = 0.45
	HG	185.4	±	84.1	183.2	±	75.1	184.6	±	4.4	
	Time	181.8	±	81.8	181.2	±	80.3				
Leg Press Max Strength (1 RM / kg body weight)	HP-LG	164.2	±	45.8	179.0 ^g	±	50.9				T = 0.001
	HP-HG	164.1	±	51.2	181.6 ^g	±	54.2				D = 0.10
	HC-LG	158.4	±	53.5	174.3 ^g	±	58.3				G = 0.90
	HC-HG	158.2	±	46.8	169.9 ^g	±	50.3				D x G = 0.67
	HP	164.1	±	48.1	180.1 ^g	±	52.3	172.2	±	2.7	T x D = 0.32
	HC	158.4	±	50.8	172.5 ^g	±	55.2	165.2	±	3.2	T x G = 0.77
	LG	161.7	±	49.3	176.9 ^g	±	54.3	169.0	±	2.7	T x D x G = 0.15
	HG	161.6	±	49.4	176.8 ^g	±	52.9	168.5	±	3.2	
	Time	161.7	±	49.3	176.8 [*]	±	53.6				
Leg Press Lift Volume (kg)	HP-LG	1815.4	±	927.6	1908.1	±	990.1				T = 0.15
	HP-HG	1830.4	±	1024.4	1967.1	±	1189.7				D = 0.003
	HC-LG	1658.1	±	867.0	1580.0 ^c	±	896.5				G = 0.36
	HC-HG	1681.2	±	864.3	1749.1	±	904.4				D x G = 0.68
	HP	1821.8	±	968.3	1933.1 ^g	±	1077.9	1880.2 [†]	±	47.1	T x D = 0.11
	HC	1667.3	±	864.3	1647.4 ^a	±	901.7	1667.1	±	54.8	T x G = 0.21
	LG	1746.2	±	903.5	1763.7	±	962.7	1740.4	±	46.2	T x D x G = 0.50
	HG	1768.5	±	962.2	1876.7	±	1083.9	1806.9	±	55.5	
	Time	1755.4	±	927.5	1810.3	±	1015.1				
Peak VO₂ (mL/kg/min)	HP-LG	20.7	±	4.9	22.8 ^g	±	4.7				T = 0.001
	HP-HG	19.1	±	3.7	20.8 ^{c,g}	±	3.9				D = 0.020
	HC-LG	20.9	±	4.4	23.9 ^{c,g}	±	4.9				G = 0.001
	HC-HG	19.8	±	3.8	21.8 ^{f,g}	±	4.2				D x G = 0.85
	HP	20.0	±	4.5	21.9 ^g	±	4.5	20.9 [†]	±	0.22	T x D = 0.026
	HC	20.5	±	4.2	23.1 ^{a,g}	±	4.7	21.6	±	0.25	T x G = 0.014
	LG	20.8	±	4.7	23.3 ^{b,g}	±	4.8	22.1	±	0.21	T x D x G = 0.28
	HG	19.4	±	3.8	21.3 ^g	±	4.1	20.4 [‡]	±	0.25	
	Time	20.2	±	4.4	22.4 [*]	±	4.6				

HP = higher protein, HC = higher carbohydrate, LG = low glucose, HG = high glucose, T = time effect, D = diet effect, G = glucose risk factor effect, D x G = diet by glucose risk factor effect, T x D = time by diet effect, T x G = time by glucose risk factor effect, T x D x G = time by diet by glucose risk factor effect.

Strength values are means ± standard deviations (except group means are means ± standard error) from 196 participants in the HP-LG group, 144 in the HP-HG group, 154 in the HC-LG group, 102 in the HC-HG group, 340 in the HP total group, 256 in the HC total group, 350 in the LG group, 246 in the HG group, and 596 participants total. Peak VO₂ values are means ± standard deviations (except group means are means ± standard error) from 214 participants in the HP-LG group, 157 in the HP-HG group, 176 in the HC-LG group, 116 in the HC-HG group, 371 in the HP total group, 292 in the HC total group, 390 in the LG group, 273 in the HG group, and 663 participants total.

* Significantly different than baseline, p < 0.05 (univariate). † Significant diet effect, p < 0.05 (univariate). ‡ Significant glucose effect, p < 0.05 (univariate). ^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than LG group, p < 0.05 (post hoc LSD). ^c Significantly different than HP-LG group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-HG group, p < 0.05 (post hoc LSD). ^f Significantly different than HC-LG group, p < 0.05 (post hoc LSD). ^g Significantly different than baseline, p < 0.05 (post hoc LSD).

Table E.8: Changes in SF36 - Quality of Life Inventory Values After Ten Weeks of Exercise and Dietary Intervention Based on Glucose Status

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Physical Functioning	HP-LG	77.4	± 32.7	84.6 ^a	± 24.9				T = 0.001
	HP-HG	73.6	± 25.8	77.2 ^c	± 24.1				D = 0.62
	HC-LG	77.2	± 24.4	82.8 ^b	± 21.5				G = 0.20
	HC-HG	75.1	± 15.5	82.8 ^b	± 18.3				D x G = 0.38
	HP	75.7	± 29.7	81.3 ^b	± 24.7	78.2	± 1.76		T x D = 0.65
	HC	76.4	± 21.2	82.8 ^b	± 20.2	79.5	± 1.88		T x G = 0.79
	LG	77.3	± 28.8	83.7 ^b	± 23.2	80.5	± 1.68		T x D x G = 0.30
	HG	74.2	± 21.8	79.7 ^b	± 21.8	77.2	± 1.95		
	Time	76.0	± 26.0	82.0*	± 22.7				
Role Physical	HP-LG	152.6	± 139.9	165.0	± 154.3				T = 0.43
	HP-HG	129.6	± 134.5	124.1	± 137.8				D = 0.001
	HC-LG	253.2	± 146.0	259.7 ^c	± 146.1				G = 0.16
	HC-HG	241.8	± 137.6	241.2 ^d	± 149.3				D x G = 0.61
	HP	142.2	± 137.5	146.4	± 148.0	142.8†	± 11.36		T x D = 0.95
	HC	248.6	± 142.3	252.3 ^a	± 147.1	249.0	± 12.16		T x G = 0.13
	LG	202.6	± 151.2	212.1	± 157.2	207.6	± 10.86		T x D x G = 0.51
	HG	179.4	± 146.4	176.1	± 153.9	184.2	± 12.61		
	Time	192.6	± 149.4	196.6	± 156.5				
Bodily Pain	HP-LG	60.8	± 19.0	64.6 ^b	± 20.2				T = 0.009
	HP-HG	57.0	± 18.9	61.4 ^b	± 21.3				D = 0.50
	HC-LG	62.7	± 21.4	63.9	± 21.4				G = 0.25
	HC-HG	60.9	± 21.6	62.4	± 22.2				D x G = 0.69
	HP	59.1	± 19.0	63.1 ^b	± 20.7	60.9	± 1.53		T x D = 0.18
	HC	62.0	± 21.4	63.3	± 21.6	62.5	± 1.64		T x G = 0.81
	LG	61.8	± 20.2	64.3	± 20.8	63.0	± 1.47		T x D x G = 0.94
	HG	58.7	± 20.2	61.8	± 21.6	60.4	± 1.70		
	Time	60.5	± 20.2	63.2*	± 21.1				
General Health	HP-LG	52.8	± 28.3	59.2 ^b	± 28.6				T = 0.001
	HP-HG	47.4	± 28.8	49.5 ^c	± 28.0				D = 0.001
	HC-LG	67.6	± 21.4	70.7 ^{c,g}	± 20.9				G = 0.03
	HC-HG	61.4	± 23.4	65.0 ^{d,g}	± 23.7				D x G = 0.79
	HP	50.4	± 28.6	54.8 ^b	± 28.6	52.2†	± 2.03		T x D = 0.53
	HC	65.1	± 22.3	68.4 ^{a,g}	± 22.1	66.2	± 2.17		T x G = 0.19
	LG	60.2	± 26.1	64.9 ^{b,g}	± 25.6	62.5	± 1.94		T x D x G = 0.10
	HG	53.6	± 27.4	56.4 ^b	± 27.2	55.8‡	± 2.26		
	Time	57.3	± 26.8	61.2*	± 26.6				
Vital	HP-LG	38.7	± 22.0	47.7 ^b	± 23.9				T = 0.001
	HP-HG	44.3	± 64.1	50.8 ^b	± 65.7				D = 0.09
	HC-LG	53.1	± 17.6	56.0	± 17.5				G = 0.97
	HC-HG	46.3	± 19.1	54.9 ^b	± 20.1				D x G = 0.33
	HP	41.3	± 46.1	49.1 ^b	± 47.5	45.4	± 2.90		T x D = 0.24
	HC	50.4	± 18.5	55.5 ^b	± 18.6	52.6	± 3.10		T x G = 0.37
	LG	45.9	± 21.2	51.8 ^b	± 21.3	48.9	± 2.77		T x D x G = 0.020
	HG	45.2	± 49.3	52.6 ^b	± 50.7	49.1	± 3.22		
	Time	45.6	± 36.0	52.2*	± 36.8				

Table E.8: Continued

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Social Functioning	HP-LG	48.1	± 24.8	54.7 ^g	± 27.3				T = 0.018
	HP-HG	44.0	± 26.8	45.8 ^c	± 25.4				D = 0.001
	HC-LG	58.8	± 23.4	60.4	± 23.4				G = 0.27
	HC-HG	60.0	± 27.2	59.6 ^d	± 26.3				D x G = 0.24
	HP	46.2	± 25.7	50.7 ^g	± 26.7	48.2 [†]	± 1.95	T x D = 0.08	
	HC	59.3	± 24.9	60.1 ^a	± 24.5	59.7	± 2.09	T x G = 0.10	
	LG	53.4	± 24.6	57.6 ^g	± 25.5	55.5	± 1.87	T x D x G = 0.51	
	HG	51.1	± 28.1	51.9	± 26.6	52.4	± 2.17		
Time	52.4	± 26.1	55.2 [*]	± 26.1					
Role Emotional	HP-LG	213.2	± 126.3	240.8	± 143.7				T = 0.64
	HP-HG	224.4	± 132.2	236.0	± 137.7				D = 0.001
	HC-LG	285.9	± 130.3	287.5 ^c	± 130.8				G = 0.50
	HC-HG	317.4	± 255.3	292.1 ^d	± 139.7				D x G = 0.64
	HP	218.3	± 128.7	238.6	± 140.6	228.6 [†]	± 10.85	T x D = 0.06	
	HC	298.6	± 190.3	289.4 ^a	± 133.9	295.7	± 11.61	T x G = 0.19	
	LG	249.3	± 133.0	264.0	± 139.0	256.8	± 10.37	T x D x G = 0.74	
	HG	265.7	± 201.1	260.9	± 140.8	267.5	± 12.04		
Time	256.3	± 165.6	262.7	± 139.6					
Mental Health	HP-LG	56.7	± 15.7	67.8 ^g	± 14.7				T = 0.001
	HP-HG	56.3	± 15.4	61.9 ^{c,g}	± 15.6				D = 0.036
	HC-LG	61.5	± 14.0	67.6 ^g	± 12.4				G = 0.12
	HC-HG	58.4	± 14.8	67.7 ^{d,g}	± 11.8				D x G = 0.57
	HP	56.5	± 15.5	65.1 ^g	± 15.3	60.7 [†]	± 1.01	T x D = 0.71	
	HC	60.3	± 14.4	67.6 ^g	± 12.1	63.8	± 1.08	T x G = 0.54	
	LG	59.1	± 15.1	67.7 ^g	± 13.6	63.4	± 0.96	T x D x G = 0.014	
	HG	57.2	± 15.1	64.5 ^g	± 14.3	61.1	± 1.12		
Time	58.3	± 15.1	66.3 [*]	± 13.9					
HP = higher protein, HC = higher carbohydrate, LG = low glucose, HG = high glucose, T = time effect, D = diet effect, G = glucose risk factor effect, D x G = diet by glucose risk factor effect, T x D = time by diet effect, T x G = time by glucose risk factor effect, T x D x G = time by diet by glucose risk factor effect. Values are means ± standard deviations (except group means are means ± standard error) from 83 participants in the HP-LG group, 69 in the HP-HG group, 82 in the HC-LG group, 55 in the HC-HG group, 152 in the HP total group, 137 in the HC total group, 165 in the LG group, 124 in the HG group, and 289 participants total. * Significantly different than baseline, p < 0.05 (univariate). † Significant diet effect, p < 0.05 (univariate). ‡ Significant glucose effect, p < 0.05 (univariate). ^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than LG group, p < 0.05 (post hoc LSD). ^c Significantly different than HP-LG group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-HG group, p < 0.05 (post hoc LSD). ^e Significantly different than HC-LG group, p < 0.05 (post hoc LSD). ^f Significantly different than baseline, p < 0.05 (post hoc LSD).									

Table E.9: Changes in Body Image Evaluation After Ten Weeks of Exercise and Dietary Intervention Based on Glucose Status

Variable	Group	Baseline		10 Weeks		Group (SEM)			P-level
Appearance Evaluation	HP-LG	2.4	± 0.7	2.8 ^g	± 0.7				T = 0.001
	HP-HG	2.4	± 0.6	2.7 ^g	± 0.7				D = 0.54
	HC-LG	2.4	± 0.6	2.9 ^g	± 0.7				G = 0.65
	HC-HG	2.4	± 0.6	2.7 ^g	± 0.7				D x G = 0.56
	HP	2.4	± 0.6	2.7 ^g	± 0.7	2.6	± 0.04		T x D = 0.64
	HC	2.4	± 0.6	2.8 ^g	± 0.7	2.6	± 0.04		T x G = 0.08
	LG	2.4	± 0.7	2.8 ^g	± 0.7	2.6	± 0.04		T x D x G = 0.57
	HG	2.4	± 0.6	2.7 ^g	± 0.7	2.6	± 0.05		
	Time	2.4	± 0.6	2.8*	± 0.7				
Appearance Orientation	HP-LG	4.3	± 0.8	4.3	± 0.9				T = 0.31
	HP-HG	3.9	± 0.9	3.9 ^c	± 0.9				D = 0.032
	HC-LG	4.0	± 0.8	4.0 ^c	± 0.9				G = 0.002
	HC-HG	3.8	± 0.7	3.8	± 0.8				D x G = 0.28
	HP	4.1	± 0.9	4.1	± 0.9	4.1 [†]	± 0.05		T x D = 0.66
	HC	3.9	± 0.8	3.9 ^a	± 0.9	3.9	± 0.06		T x G = 0.52
	LG	4.2	± 0.8	4.1 ^b	± 0.9	4.1	± 0.05		T x D x G = 0.15
	HG	3.9	± 0.8	3.9	± 0.8	3.9 [‡]	± 0.06		
	Time	4.1	± 0.8	4.0	± 0.9				
Body Area Satisfaction	HP-LG	2.0	± 0.8	2.2 ^g	± 0.8				T = 0.001
	HP-HG	2.1	± 0.7	2.4 ^g	± 0.9				D = 0.002
	HC-LG	2.2	± 0.7	2.5 ^{c,g}	± 0.9				G = 0.18
	HC-HG	2.3	± 0.7	2.6 ^g	± 0.8				D x G = 0.69
	HP	2.0	± 0.8	2.3 ^g	± 0.8	2.2 [†]	± 0.05		T x D = 0.32
	HC	2.2	± 0.7	2.5 ^{a,g}	± 0.8	2.4	± 0.06		T x G = 0.56
	LG	2.1	± 0.8	2.4 ^g	± 0.8	2.2	± 0.05		T x D x G = 0.25
	HG	2.2	± 0.7	2.5 ^g	± 0.8	2.3	± 0.06		
	Time	2.1	± 0.7	2.4*	± 0.8				
Overweight Preoccupation	HP-LG	2.9	± 0.7	3.4 ^g	± 0.7				T = 0.001
	HP-HG	2.9	± 0.6	3.4 ^g	± 0.7				D = 0.22
	HC-LG	2.9	± 0.7	3.3 ^g	± 0.7				G = 0.31
	HC-HG	2.7	± 0.7	3.2 ^g	± 0.7				D x G = 0.29
	HP	2.9	± 0.7	3.4 ^g	± 0.7	3.1	± 0.04		T x D = 0.26
	HC	2.9	± 0.7	3.3 ^g	± 0.7	3.1	± 0.05		T x G = 0.72
	LG	2.9	± 0.7	3.4 ^g	± 0.7	3.1	± 0.04		T x D x G = 0.18
	HG	2.8	± 0.7	3.3 ^g	± 0.7	3.1	± 0.05		
	Time	2.9	± 0.7	3.3*	± 0.7				
Self-Classified Weight	HP-LG	4.1	± 0.8	4.1	± 0.6				T = 0.008
	HP-HG	4.5	± 0.5	4.2 ^g	± 0.7				D = 0.036
	HC-LG	4.2	± 0.7	4.0 ^{c,g}	± 0.7				G = 0.009
	HC-HG	4.2	± 0.7	4.1	± 0.7				D x G = 0.35
	HP	4.2	± 0.7	4.2 ^g	± 0.7	4.2 [†]	± 0.04		T x D = 0.93
	HC	4.2	± 0.7	4.0	± 0.7	4.1	± 0.04		T x G = 0.41
	LG	4.1	± 0.8	4.1	± 0.7	4.1	± 0.03		T x D x G = 0.010
	HG	4.3	± 0.6	4.2 ^g	± 0.7	4.2 [‡]	± 0.04		
	Time	4.2	± 0.7	4.1*	± 0.7				

Table E.9: Continued

Variable	Group	Baseline			10 Weeks			Group (SEM)			P-level
Rosenberg Self Esteem	HP-LG	26.2	±	4.1	26.5	±	3.8				T = 0.006
	HP-HG	25.0	±	3.6	26.0 ^g	±	3.8				D = 0.67
	HC-LG	25.8	±	4.1	26.2	±	4.2				G = 0.06
	HC-HG	25.5	±	3.7	25.5	±	3.7				D x G = 0.62
	HP	25.7	±	3.9	26.3 ^g	±	3.8	25.9	±	0.23	T x D = 0.10
	HC	25.7	±	3.9	25.9	±	4.0	25.8	±	0.26	T x G = 0.53
	LG	26.0	±	4.1	26.4	±	4.0	26.2	±	0.21	T x D x G = 0.11
	HG	25.2	±	3.7	25.8 ^g	±	3.8	25.5	±	0.27	
	Time	25.7	±	3.9	26.2 [*]	±	3.9				
Social Physique Anxiety	HP-LG	31.1	±	6.4	31.6	±	6.4				T = 0.74
	HP-HG	31.4	±	6.1	30.9	±	5.9				D = 0.82
	HC-LG	31.3	±	6.1	31.2	±	5.1				G = 0.72
	HC-HG	30.7	±	5.8	31.4	±	7.4				D x G = 0.99
	HP	31.3	±	6.3	31.4	±	6.2	31.3	±	0.33	T x D = 0.70
	HC	31.1	±	6.0	31.3	±	6.1	31.2	±	0.38	T x G = 0.95
	LG	31.2	±	6.3	31.4	±	5.9	31.3	±	0.31	T x D x G = 0.18
	HG	31.1	±	5.9	31.2	±	6.6	31.1	±	0.40	
	Time	31.2	±	6.1	31.3	±	6.2				
HP = higher protein, HC = higher carbohydrate, LG = low glucose, HG = high glucose, T = time effect, D = diet effect, G = glucose risk factor effect, D x G = diet by glucose risk factor effect, T x D = time by diet effect, T x G = time by glucose risk factor effect, T x D x G = time by diet by glucose risk factor effect. Values are means ± standard deviations (except group means are means ± standard error) from 161 participants in the HP-LG group, 95 in the HP-HG group, 120 in the HC-LG group, 75 in the HC-HG group, 256 in the HP total group, 195 in the HC total group, 281 in the LG group, 170 in the HG group, and 451 participants total. * Significantly different than baseline, p < 0.05 (univariate). † Significant diet effect, p < 0.05 (univariate). ‡ Significant glucose effect, p < 0.05 (univariate). ^a Significantly different than HP group, p < 0.05 (post hoc LSD). ^b Significantly different than LG group, p < 0.05 (post hoc LSD). ^c Significantly different than HP-LG group, p < 0.05 (post hoc LSD). ^d Significantly different than HP-HG group, p < 0.05 (post hoc LSD). ^e Significantly different than HC-LG group, p < 0.05 (post hoc LSD). ^f Significantly different than baseline, p <0.05 (post hoc LSD).											

APPENDIX F

CARBOHYDRATE INTOLERANCE QUESTIONNAIRE

Baylor University
Exercise & Sport Nutrition Laboratory

Trial: Effects of the Curve for Women Fitness and Weight Loss Program on Body Composition, Metabolism, and Exercise Capacity in Sedentary Overweight Females
Carbohydrate Tolerance Questionnaire

Group: _____

The Institute for Nutritional Science has devised a sample of questions to determine if you are carbohydrate intolerant or calorie sensitive. Please complete the following three tests to help us better determine your best method of dieting.

Test I: Symptoms of Carbohydrate Intolerance - Have you ever experienced any of the following symptoms?

- | | |
|-------------------------------|---|
| 1. _____ Insomnia | 11. _____ Lack of sex drive |
| 2. _____ Irritability | 12. _____ Fatigue and Exhaustion |
| 3. _____ Headaches | 13. _____ Overemotional crying spells |
| 4. _____ Depression | 14. _____ Leg cramps and blurred vision |
| 5. _____ Nervousness | 15. _____ Cravings for starch and sugar rich foods |
| 6. _____ Muscle pains | 16. _____ Digestive disturbances with no apparent cause |
| 7. _____ Forgetfulness | 17. _____ Rapid pulse, especially after eating certain foods |
| 8. _____ Mental confusion | 18. _____ Shortness of breath, sighing and excessive yawning |
| 9. _____ Needless worrying | 19. _____ Drowsiness, especially after meals or in mid afternoon |
| 10. _____ Antisocial behavior | 20. _____ Faintness, dizziness, cold sweats, shakiness, weak spells |

_____ Total # of "yes" answers.

_____ Total # of "no" answers.

Test II: Carbohydrate Intolerance – Respond to the following statements with either a yes or no.

1. _____ You are more than 25 pounds overweight.
2. _____ You have had a tendency to be overweight all of your life.
3. _____ You have been overweight since you were very young.
4. _____ You have a poor appetite and often skip meals.
5. _____ You have food cravings that temporarily go away when starch or sugary foods are eaten.
6. _____ There are foods that you feel you absolutely could not do without.
7. _____ Your waistline is bigger than your hips.
8. _____ Most or all of the symptoms associated with carbohydrate intolerance apply to you (Test I).

_____ Total # of "yes" answers.

_____ Total # of "no" answers.

Test III: Calorie Sensitivity – Respond to the following statements with either a yes or no.

1. _____ You had a normal body weight when younger but slowly gained weight after age 30.
2. _____ You are presently overweight but by less than 25 pounds.
3. _____ You have a normal appetite – get hungry at meal times.
4. _____ You have few, if any, food cravings.
5. _____ You have maintained the same basic eating habits all of your life.
6. _____ You eat three meals per day.
7. _____ You have gained a certain amount of extra body weight but seem to have tapered off (not continued to steadily gain more and more weight).
8. _____ You have few or none of the symptoms associated with poor carbohydrate metabolism (Test I).

_____ Total # of "yes" answers.

_____ Total # of "no" answers.